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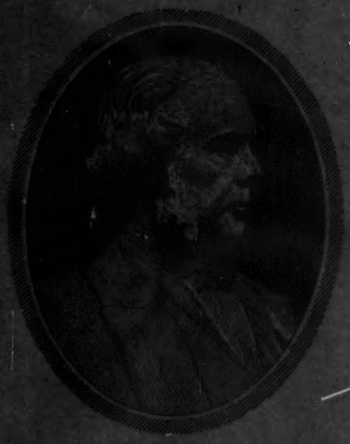

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THE
BRITISH JOURNAL OF SURGERY

VOL. XXXIV OCTOBER, 1946 No. 134

ENDOMETRIOSIS: A SURGICAL PROBLEM*

BY PROFESSOR DOUGLAS MACLEOD

THE term 'endometriosis' implies the invasion by the endometrium of tissues to which it is foreign. Of the sites where such invasion takes place, the uterus, ovaries, and the peritoneum of the pouch of Douglas are by far the most conspicuous and the literature on this part of the subject is already very large. From time to time, however, cases are recorded where the locus is situated in tissues either right outside the genital tract, as the bowel, the umbilicus, and the bladder and ureter, or in positions on the very fringe of it, such as the round ligament in the groin or the perineum.

It is my purpose this afternoon to deal mainly with extragenital endometriosis; first, because not much has been written about it; secondly, because it is of interest to all surgeons whose province is the abdomen; and thirdly, because it throws a light on the much debated question of the genesis and method of spread of endometriosis in general.

In the normal woman the characteristic mucous membrane of the uterus is strictly confined to the interior of the corpus, but in the condition known as endometriosis areas of tissue indistinguishable from uterine mucosa are found in other situations. Here they may give rise to the formation of tumours which we call endometriomata, capable of a diversity of puzzling symptoms.

The tissue of an endometrioma is remarkable in that it is subject to the same cyclical changes as the uterine mucosa in its normal situation—that is to say, proliferative and secretory phases at ordinary times, and, in the event of a pregnancy, a decidual reaction comparable with that of the gravid uterus. Like endometrium, therefore, the endometrioma menstruates; but, because it lacks the normal outlet of the uterus, it is forced to retain the products of its menstruation in the form of small intertissue aggregations or, when the ovarian follicle is affected, the 'chocolate' cyst, so characteristic of this condition. These features are evidently dependent on the presence of active ovarian tissue, since endometriosis does not occur before the child-bearing period of life and disappears spontaneously after the menopause, whether natural or artificially induced. It also tends to disappear after a pregnancy, a fact I have been able to verify by laparotomy on such cases. Pregnancy is, however, uncommon in endometriosis because of the high sterility rate with which it is associated.

In its simplest form, endometriosis arises by direct invasion (*Fig.* 144). This is exemplified by endometriosis (adenomyoma) of the wall of the uterus, where the spread is certainly by way of the intertissue spaces.

FIG. 144.—Endometriosis (adenomyosis) of the uterus. The endometrium is seen directly invading the uterine muscle.

Direct invasion, however, does not explain the appearance of endometrial tissue in situations remote from the uterus, and Sampson (1921) produced the Implantation Theory, which supposes that endometrial cells pass through the Fallopian tubes during menstruation and become implanted on the ovary and other structures in the pelvis, particularly the peritoneum in the pouch of Douglas. Local invasion of these structures follows, and cysts and tumours are produced containing areas of endometrium and larger or smaller collections of retained menstrual blood.

Iwanoff (1889) and others advanced the Serosal Theory, which holds that under certain conditions the peritoneum, because it is developed from the same structure as the uterine mucosa—namely, the splanchnopleure—has the power to undergo metaplasia and reproduce tissue resembling endometrium. During pregnancy it is common to find small moss-like tufts on the surface of the ovary, in the pouch of Douglas, and even on the peritoneal surface of the bowel, which, when scraped off and sectioned, show pure decidua.

* Being a Hunterian Lecture delivered at the Royal College of Surgeons of England, May 9, 1946.

These are probably due to metaplasia, and I think it significant that they are found in sites where it is common for endometriosis to occur. If this theory could be extended to include metaplasia of the granulosa cells of the ovary, also derived from the same embryonic source, many of the puzzling features of endometriosis would be explained. I have, in a previous paper (1934), attempted to demonstrate that these cells appear to be capable of developing an epithelium in ovaries containing endometrial cysts, and it is possible that the 'chocolate' cyst is formed in this way. It must, however, be admitted that proof of metaplasia is lacking, and that once one admits metaplasia, anything can be explained.

Lastly, there is the view that endometriosis arises by lymphatic spread from the uterus, in much the same way as a malignant neoplasm metastasizes by means of the lymphatic system. Strong support for this view has been afforded by the discovery of islands of endometrial tissue in the lymph-glands and even in the lymphatic ducts themselves.

Before discussing extragenital endometriomata it is necessary to give a brief account of the more common manifestations of the disease. In the uterus it gives rise to a tumour very closely resembling a fibroid both in superficial appearance and symptoms—that is, it forms a protuberance and is accompanied by more or less excessive menstrual bleeding. There is, however, a difference; fibroids generally are painless tumours, but uterine endometriomata give rise to severe pain at the periods. This is not to be wondered at since at every menstrual epoch numerous isolated collections of blood are being formed in the dense tissue of the tumour. This severe pain at the time of the periods is characteristic of endometriosis in most of the other situations where it occurs.

Endometriosis when it affects the ovary usually produces what is called the 'chocolate' or 'tarry' cyst (*Fig.* 145). Careful microscopical examination

FIG. 145.—Endometrial cyst of the ovary.

of the wall of these cysts discloses in them tissue indistinguishable from endometrium. There can be no doubt at all that such cysts are in fact the ovarian follicles distended by retained menstrual blood. They are frequently multiple in the ovary and may on occasion attain a large size. Their characteristic symptom is the appearance of increasing pain at each monthly period in a patient who has had previously no pain at all. It is a feature of these cysts that they tend to perforate. If this is gradual, the point of imminent perforation becomes adherent to adjacent structures, usually the posterior layer of the broad ligament, and these in turn are invaded by endometrial tissue. If, on the other hand, the perforation is sudden, the contents of the cyst leak into the peritoneal cavity, giving rise to an acute abdominal emergency. A knowledge of these cysts is therefore of importance to the general surgeon, for the sudden rupture of a chocolate cyst causes severe peritoneal irritation and symptoms closely resembling those of acute appendicitis. Gradual perforation is unlikely to cause so dramatic a picture, but even here a chronic

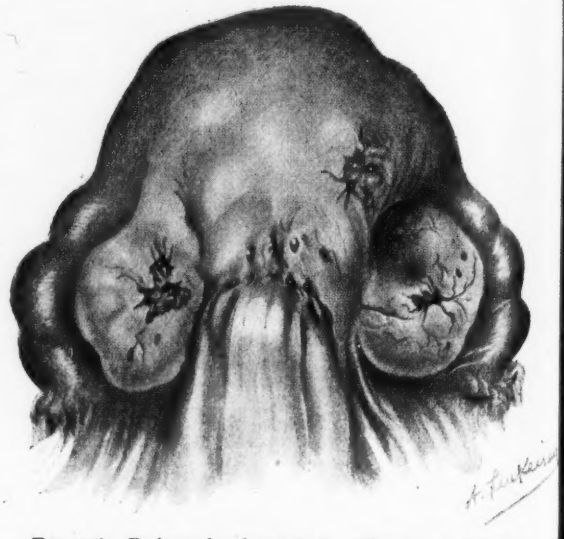


FIG. 146.—Peritoneal endometriosis. The rectum is drawn up and adherent to the cervix, obliterating the pouch of Douglas—a characteristic feature.

appendix may be suspected, and the organ removed without alleviation of the symptoms. In the treatment of ovarian endometriosis it is important to remember that in younger women the main principle must always be to preserve ovarian tissue, and nearly always the cyst, irrespective of its size, can be enucleated from out the ovary. There is another form of endometriosis affecting the ovary which exhibits small superficial areas resembling blood blisters on the surface (*Fig.* 146). It may or may not be associated with similar deposits on the pelvic peritoneum and is essentially part of a peritoneal endometriosis. The peritoneum will show either discrete nodules, some obviously blood-stained, or a diffuse thickening which becomes very adherent and rigid. The front and back wall of Douglas's pouch and the lower part of the posterior face of the broad ligament are the parts most commonly affected, but one case at least is on record where a small endometrioma was found on the under surface of the diaphragm. The rectum becomes characteristically drawn up and, adhering to the cervix, obliterates the pouch of Douglas. Curiously enough, in this situation the characteristic pain is often absent. From the pouch of Douglas extension may occur into the vaginal vault or into the rectovaginal septum—in the latter case even invading the rectum.

I pass on now to the rarer situations in which endometriomata are found.

Round Ligament.—Seeing that the round ligament is in direct continuity with the uterus, it might be thought that endometriosis in this

situation would be common, but such cases are rare, and I have only seen one myself, but Mr. Zachary Cope has kindly allowed me to quote a recent case of his. The patient was admitted as a case of strangulated hernia. She complained of a painful swelling in the right groin which varied in size and which she had had for five years. Two years prior to its first appearance she had a hysterectomy for fibroids. Examination showed a firm, tender mass about 1¼ in. in diameter in the region of the right external inguinal ring. At operation it was found to have no capsule, but there were a number of small blood spaces on the cut surface. Section confirmed that it was an endometrioma. The features of the one case of which I have personal experience were practically identical with those of Mr. Cope's patient, and I am also acquainted with another where the swelling was situated well below the level of the external abdominal ring. It was excised and found to be an endometrioma and—this is the most interesting part of the case—a recurrence took place 18 months later lower down still in the substance of the labia majora. This also was excised and proved to be endometriomatous.

The Perineum.—Cases are recorded of endometriosis affecting the lower part of the genital tract—that is, the cervix, the vagina below the

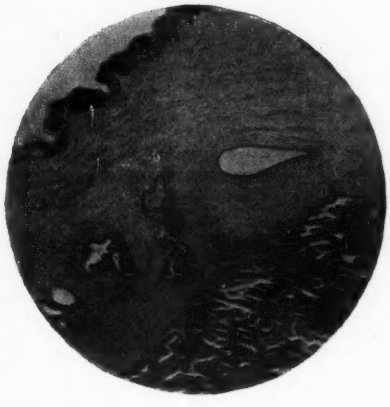


FIG. 147.—Endometrioma of the perineum. (*Mr. J. White's case.*)

fornix, and the perineum—but they are rare; and when one considers the many opportunities that are afforded for endometrial tissue to implant, after labour or after repair operations preceded by a curettage, it is rather surprising that this is so; in fact, probably not more than a dozen cases of perineal endometriosis have been described in the literature.

I have been furnished with the details of such a case. She had had one child born in 1935, when the perineum was torn and sutured. A year later she noticed itching in the perineum where a small lump could be felt. Later this began to bleed two days before a menstrual period and after trauma. When seen three years later there was a raised purple warty tumour of fleshy consistency and about the size of a walnut situated in the perineum. It was noticed that the rectovaginal septum above the tumour was normal, and that there was no apparent abnormality of the pelvic organs (*Fig.* 147). Excision and microscopy confirmed that it was an endometrioma.

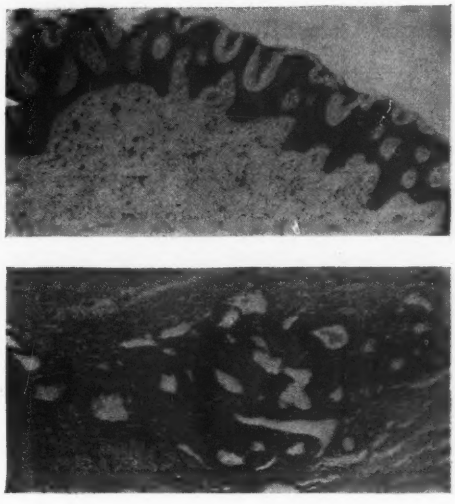


FIG. 148.—Endometrioma of the umbilicus.

The Umbilicus.—Rather fewer than 50 cases of umbilical endometriosis have been reported, and most of these have occurred in the fourth decade of life. It appears as a small lump in the umbilicus, which enlarges and becomes tender at the time of the menstrual period. In some instances bleeding occurred externally from the tumour (*Fig.* 148). Pelvic endometriosis is often present as well. Treatment is by simple excision, but the coexistence of pelvic endometriosis will naturally call for separate measures.

FIG. 149.—Endometrioma of the bladder. Premenstrual appearance on cystoscopic examination. (*Mr. R. B. Phillips's case—reproduced from the "Journal of Obstetrics and Gynæcology of the British Empire", by courtesy of the Editors.*)

Bladder.—Endometrioma of the bladder is a very rare, but important, disease. L. R. Reynolds (1939) collected 34 cases from the literature and added 1 of his own. The bladder may be involved in two ways: either by direct extension from endometriosis of other pelvic organs, such as the uterus or ovary; or it may appear to arise *de novo*

in the bladder wall itself. The posterior wall of the bladder—in the neighbourhood of the trigone—is usually affected and the ureteric orifices may be involved. The tumour primarily is confined to the muscular wall, but later may invade the epithelial lining. It is not encapsulated and at operation may be mistaken for a carcinoma. In a case reported by Phillips (1934), cystoscopy revealed an ulcerating polypoid growth, leading to a diagnosis of carcinoma, and it was only after a transurethral biopsy had been done that its true nature was recognized. The appearance on cystoscopic examination depends on whether the mucosa is encroached upon. If not, the tumour appears as a rather sharply defined area of congestion, but when the epithelium is invaded, there will be seen grape-like cysts and what has been described by Phillips as "blue folds of œdema" (*Fig.* 149).

The symptoms are usually characteristic. There are cyclical attacks of frequency of micturition, dysuria, and suprapubic pain. If the epithelium is involved, hæmaturia will occur at the time of the menstrual period. Examination of the anterior fornix will show a tender mass in the uterovesical fold and cystoscopy supported by biopsy will clinch the diagnosis.

Treatment depends on (1) the age of the patient and (2) whether the ureteric orifices are involved. As with endometriosis elsewhere in patients in the neighbourhood of 40 years of age, castration by irradiation will be followed by regression of the tumour and disappearance of symptoms in most cases.

In young patients radical resection may be preferable. If the ureteric orifice is involved partial cystectomy and reimplantation of the ureter may be necessary.

Occasionally the condition recurs after local excision and even after castration, and in such cases partial cystectomy is the only course.

Ureter.—Only 2 cases of primary ureteric endometrioma have been reported, but the ureter may be involved secondarily in endometriosis of the cervix, the pouch of Douglas, the rectovaginal septum, and the ovary if the latter is adherent to the side wall of the pelvis. This occurs far more commonly than is supposed and is a most important factor in some cases of otherwise unexplained recurrent pyelitis, of which I have seen several. Treatment follows along the same principles as before—in young patients excision of the affected part of the ureter with reimplantation into the bladder being required when the duct is deeply affected.

Laparotomy Scar.—Endometrioma occurring in a laparotomy scar is not rare, and affords the strongest evidence that direct implantation of endometrium can occur (*Fig.* 150). Many of these cases follow operations in which the cavity of the uterus has been opened, such as Cæsarean section, or during myomectomy, or after abdominal curettage. They may also follow salpingectomy and the operation of ventrofixation. Sampson in a recent paper (1945) studied 17 cases, and in 16 the condition had followed a salpingectomy. In his experience the shortest interval between the operation and the appearance of the tumour was two years, the longest seventeen years. Mr. Victor Bonney tells me he has had 6 cases of his own, 5 of which followed abdominal curettage. In none of his cases was the parietal peritoneum involved, nor was there any evidence of endometriosis in the pelvis. The tumours in these cases were undoubtedly the result of direct implantation of endometrium in the abdominal wall, and it is worthy of note that since he used rubber sheeting to protect the exposed tissues of the abdominal wall, he has had no further cases. If laparotomy and inspection of the pelvis indicates that the condition is an isolated one, local excision is enough, but in cases where there is evidence of direct continuity with the uterus or appendages hysterectomy will probably be required.

FIG. 150.—Endometrioma in a laparotomy scar.

The Bowel.—Endometriosis affecting the intestinal tract is the sphere likely to be of most interest and concern to the abdominal surgeon. The small and large intestines may both be the site of endometriosis. In some cases the bowel is found attached to an area of endometriosis in the genital organs, direct invasion occurring in this way. Especially is this likely in the case of the sigmoid colon, while the rectum is prone to be invaded directly from endometriosis in the rectovaginal septum. The appendix, too, may be similarly involved, and Sampson has shown that this is most likely when that organ dips into the pelvic cavity.

It is, however, most important to remember that the bowel may be the site of endometriosis without the pelvic organs being affected, and modifications in the clinical picture are to be expected accordingly.

The Small Intestine.—The symptoms of intestinal endometriosis are subject to minor variations according to the segment of bowel concerned. In the case of the small bowel the patient will give a history of increasing pain with attacks of vomiting, distension, and constipation which coincide with the monthly periods. Here are two illustrative cases :—

The first was a patient aged 44, who had an attack of severe pain in August, 1940, which was diagnosed as an appendicitis. She was seen six months later complaining of severe abdominal pain, and she had

all the symptoms of intestinal obstruction. At operation the small bowel was found to be grossly distended and the colon collapsed. There were two tight strictures, 4 in. and 6 in. proximal to the ileocæcal valve, closely resembling a carcinoma. Resection of the affected bowel was performed and the patient made an excellent recovery. Microscopy showed that the strictures were due to endometriomatous deposits.

The second was a patient, aged 37, who had had attacks of pain in the right lower abdomen for some time, recently becoming more frequent and finally very severe. An acute appendicitis was diagnosed. At operation, however, the appendix was found to be normal,

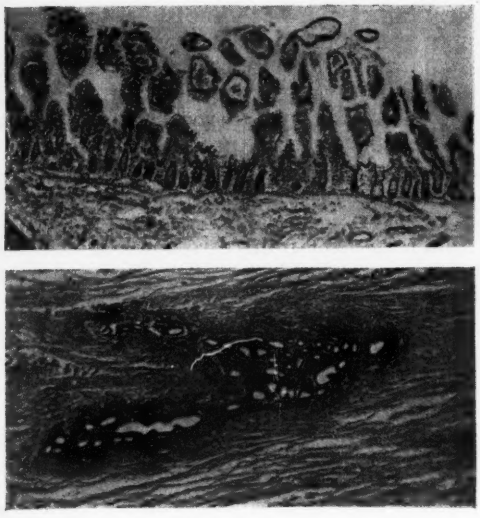


FIG. 151.—Endometrioma of the small intestine. (*Dr. E. C. Atkinson's case.*)

but 18 in. from the ileocæcal valve the bowel was much puckered over a tumour in its wall about the size of a small walnut and very hard. As the condition was thought to be a carcinoma, resection was performed and the patient made an uneventful recovery. The pathologist reported an adenomyoma of uterine origin (*Fig.* 151).

I would like to emphasize some points common to these two cases. In both, the symptoms were most noticeable at the time of menstruation and were becoming more and more severe. Both at first were considered to be cases of appendicitis, though in the former case signs of intestinal obstruction eventually supervened. At operation both were considered to be carcinoma and were treated with resection. On opening the gut, however, after it had been resected, it was noticed that there was no involvement of the mucosa—a significant observation.

Sigmoid Colon.—Several cases of endometrioma of the sigmoid colon have been reported. Complete intestinal obstruction in this situation is very rare. Occasionally the patient may pass blood per rectum during a menstrual period, but this is uncommon. The mucosa is rarely invaded and sigmoidoscopy reveals little, except that its performance causes considerable pain and may be difficult owing to surrounding adhesions. Any degree of stricture may be found. I will describe a few cases to illustrate this condition :—

The first, a patient aged 40, complained of abdominal pain for a week before each period and constipation was very marked at this time. At operation endometriosis was seen involving the posterior uterine wall, both ovaries, and the pouch of Douglas. There was also a

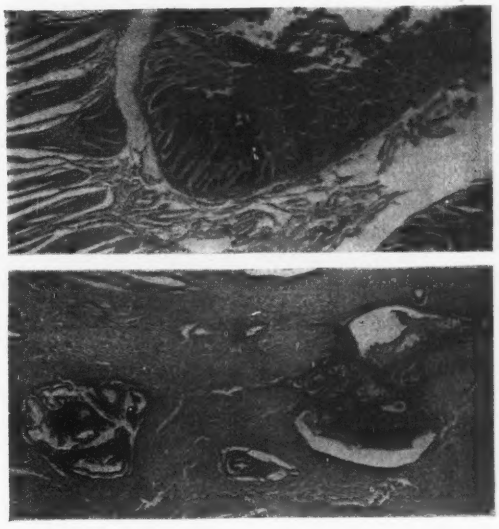


FIG. 152.—Endometrioma of the sigmoid colon. (*Mr. J. B. Blaikley's case.*)

stricture in the middle of the pelvic colon which the surgeon correctly diagnosed as an endometrioma (*Fig.* 152). He performed hysterectomy, removed both ovaries, and resected the involved pelvic loop—performing a Paul's operation. The bowel in this case was completely encircled and it was not possible to get the tip of the little finger through the constricting ring. The mucous membrane was not involved and moved freely over the constriction.

The second, a young woman aged 30, complained of severe premenstrual pain of recent origin in the left ovarian region. Clinical findings made me suspect a left ovarian endometrial cyst. At laparotomy I was surprised to find no evidence of genital endometriosis, but so typical was her history that I explored the bowel, and at the lower end of the sigmoid I found a tumour the size of an olive showing typical blood-spots. I was able to perform a local excision, avoiding a resection which would have been difficult at this level.

The third occurred in a woman, aged 50, complaining of pain in the left iliac fossa which was exacerbated at the menstrual period. She had an endometrioma of the left ovary which had invaded the sigmoid colon. Both ovaries were removed and she is now free from symptoms.

The fourth was a woman, aged 51, with multiple fibroids, but as she had complained of severe left-sided pain, constipation, and distension, her doctor suspected an intestinal growth. He asked me to explore the bowel when I performed the hysterectomy. This I did, and in the pelvic colon I found a tumour the size of a walnut, showing the typical blood-cysts. As she was 51, I did no more than remove both ovaries. Shrinkage must have occurred rapidly, for within three weeks she told me she was now free from pain.

Rectum.—As endometrium of the pouch of Douglas is common, some involvement of the rectum may be said to be a fairly frequent occurrence. The degree of involvement varies from a mild

condition to an extensive involvement of the bowel, especially in cases of advanced endometriosis of the rectovaginal septum; in fact, cases have been diagnosed as carcinoma and treated by abdomino-perineal excision. The symptoms of pain on defæcation, with bleeding, and a mass felt per rectum, may well lead the surgeon to make a diagnosis of rectal cancer, but if a proctoscope is passed the mucosa appears to be unaffected. The condition may also occur higher up in the bowel and the tumour may appear as a small lump in the rectal wall or may even project into its lumen as a polypus. The following two cases illustrate this condition :—

The first was a patient who complained of rectal bleeding. On examination a sessile tumour on the anterior wall was found just in reach of the finger. Under anæsthesia the tumour was drawn down and it was seen to be covered with healthy mucous membrane. This was incised and the tumour dissected out and removed for biopsy. At the same time, it was found that the base of the tumour involved the pelvic peritoneum and removal had caused an opening into the peritoneal cavity. A second tumour higher up was not accessible for removal per rectum. The abdomen was therefore opened and extensive pelvic endometriosis was revealed. Further treatment was limited to the induction of the menopause by X rays. She reported three years later, again complaining of rectal bleeding. I was asked to see her and found she had been taking stilbœstrol for some months, which had, presumably, re-established the condition of endometriosis and consequently bleeding was occurring from the remaining polypus. At all events, when she was taken off stilbœstrol the bleeding ceased.

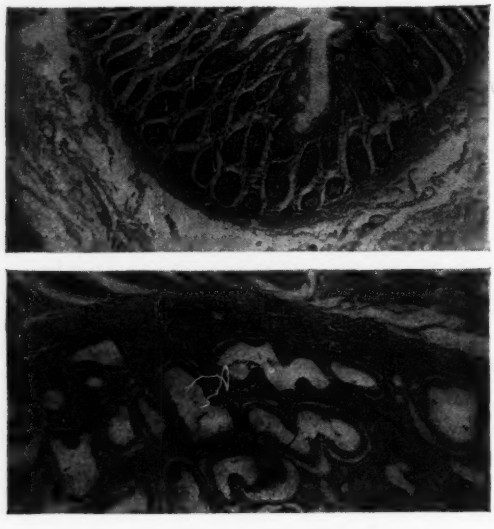


FIG. 153.—Endometrioma of the rectum.

The other case was of a different nature. The patient, aged 34, had been transferred from another hospital where laparotomy had revealed a tumour of the rectocolic junction. She complained of rectal bleeding at the menstrual periods and dysmenorrhœa of some months' duration. Examination showed a mass in the pelvis and extensive thickening in the anterior rectal wall, and a diagnosis of rectal carcinoma was made. An abdominoperineal excision was performed and the patient made an excellent recovery (*Fig.* 153). Histological examination of the growth showed an endometrioma. She remained symptom-free for about eighteen months, when she returned complaining of abdominal pain. The uterus was found to be retroverted and fixed, and a recurrence of endometriosis was thought to be sufficiently probable to warrant a small dose of intra-uterine radium. Ten months later she became pregnant and was delivered by Cæsarean section.

I saw her at the end of 1945. She is now 51 and has seen no periods for three years. Pelvic examination revealed no evidence of endometriosis and she is well and free from symptoms.

From a study of the previous cases of intestinal endometriosis it will be observed that it may be extremely difficult, if not impossible, to differentiate endometriosis from carcinoma when the abdomen is opened. The main points that may be helpful in distinguishing the two are: first, the association of pelvic endometriosis; secondly, the presence of blood-cysts in the tumour, if visible; and thirdly, for the size of the growth, secondary involvement of the glands in endometriosis is either absent or, if present, minimal. Nevertheless, in many cases differentiation from carcinoma at the time of operation is not possible with any degree of certainty, and in all cases in which there is doubt resection of the affected loop is the proper course. This applies, too, to all young patients even when the diagnosis is *not* in doubt if the condition is too extensive for local excision.

It is only possible to modify treatment in undoubted cases of endometriosis in patients over 40 where induction of an artificial menopause by oöphorectomy or radiation will avoid the need for more extensive and dangerous procedures. It may even be called for in young subjects where the pelvirectal junction or rectovaginal septum is so extensively involved that abdomino-perineal resection is the only operative procedure capable of removing the growth.

THE GENESIS AND METHOD OF SPREAD OF ENDOMETRIOSIS

I have postponed this discussion to the last because the occurrence of endometrioma outside the genital area has an important bearing on the subject.

The phenomenon of endometriosis is unique in this respect, that a whole normal adult tissue—not a single type of cell—transplants itself and grows in tissues foreign to it. Throughout active sexual life the endometrium is renewed every month, with the result that it never grows old; in fact, it is a 'Peter Pan' amongst tissues, perpetually retaining that vigorous growth propensity which makes it peculiarly suitable for tissue culture. In proof of this, twelve years ago, in this College, at Mr. Victor Bonney's suggestion, human endometrium was grown successfully *in vitro*.

With this knowledge, the occurrence of endometriomata in laparotomy scars by direct implantation becomes an event which one would expect occasionally to happen; and, further, one might anticipate that at times the endometrium would invade the uterine muscle that underlies it. What, in the first instance, tips the balance between the growth power of the endometrium and the resistance

of the muscle, is unknown to us, but it is certain that in uterine endometriosis invasion occurs along the intertissue spaces—a lymphatic permeation, in fact—the invaded tissues reacting by hypertrophy and fibrosis until a definite, though unencapsulated, tumour mass is formed. In the case of endometriosis of the ovaries and pelvic peritoneum the most popular explanation is the 'spill' theory of Sampson (1921), and as far as surface endometriosis is concerned, this is adequate. A difficulty arises in the case of the endometrial cysts of the ovary (chocolate cysts) which, it is to be remembered, are, of all types of extra-uterine endometriosis, by far the most common. They are sited in the ovarian follicles, often quite deeply embedded in the ovary and enucleable from it. Fragments of endometrium escaping from the tube could only find their way into a follicle after dehiscence, whereas many of the follicles affected have almost certainly never dehisced. The serosal theory is also incapable of explaining their occurrence, unless it is postulated that the follicular cells themselves can undergo such metaplasia. As yet, there is no proof of this.

There remains the Lymphatic Theory. The ovary is in direct lymphatic communication with the uterus and being an 'end-organ' is frequently the site of secondary carcinomatous deposits, especially when the primary growth is in the uterus. The rectum and rectocolic junction, the bladder, and the lower end of the ureter are so close to the uterus that no great length along the intertissue spaces has to be traversed. The appendix, colon, and small intestine are farther afield, but they are in communication through their mesenteries with the retroperitoneal tissue, of which the parametrium forms a part. Cases have been published where endometriomatous deposits have been found in the inguinal, pelvic, and mesenteric glands. They have even been found in the lymphatic ducts themselves —this is direct proof of lymphatic conduction.

The occurrence of umbilical endometriosis cannot be explained by any theory other than that of lymphatic spread, and in this connexion I would remind the reader of the work of Sampson Handley (1910), who showed in his study of the "Surgery of the Lymphatic System" that the umbilicus is richly supplied with lymphatics and that secondary deposits may occur there when intraperitoneal carcinomatous cells are implanted on the anterior parietal peritoneum. The presence of these metastases, he asserts, is proof that cancer cells have obtained access to the peritoneal cavity. It is noteworthy that in cases of umbilical endometriosis, obvious pelvic endometriosis frequently coexists.

It appears, therefore, from a survey of all the facts at our disposal that endometriomata wherever they occur are the result of the endometrium taking upon itself lymphatic permeation. The occurrence of 'tubal spill' cannot be absolutely disproved, but when we have an hypothesis adequately covering all the known facts it seems unnecessary to put forward a second one which only accounts for a few of them. Moreover, allowing that spill occurs, we still have to invoke lymphatic permeation to account for the occurrence of endometriomata in positions remote from the peritoneum.

EPILOGUE

In the foregoing I have tried to cover the main features of endometriosis as it is likely to concern the general surgeon as much as the gynæcologist.

I have indicated the various syndromes that may result from its extension beyond the genital tract, and I have referred to the theories that explain, or attempt to explain, the manner of its extension.

There remains but one issue still to be discussed, and I will make it the epilogue to my subject, even though it means that I must end on a note of interrogation. It is this: What place in the pathological gallery should be assigned to the picture of endometriosis? Where does it properly belong in our catalogue of diseases, that it may be seen in its true perspective in relation to other pathological processes whose position is more assured?

Clearly it is of the order of a growth, and an abnormal growth at that. Is it malignant, or is it benign?

In the vigour of its growth, in its power of invasion, and in its tendency to metastasize, it undoubtedly resembles a malignant growth. But the normal endometrium has the same vigour even outside its natural habitat, as was shown by successful tissue culture.

Further, the tissue of an endometrioma, whatever it may be, is under the same rigorous functional control as the normal endometrium—namely, that of ovarian activity. And even in its metastases, the extent of its invasion seems to be limited. Where in the sphere of malignancy is there anything comparable with this? On the other hand, we are not justified in describing the endometrioma as a benign growth and there letting the matter rest. There is much more to it than that. For instance, we have only considered the part played by the ovary in controlling the *behaviour* of endometriomata. Is it possible that some abnormality of ovarian or related endocrine function may be responsible for its *occurrence*? This view recalls the analogous theories advanced by workers on cancer, seeking to incriminate the endocrine system as the responsible agent in the production of malignant growths.

However this may be, it seems that endometriosis must be accorded a position somewhere between benign and malignant growths, two separate states of which the true relation has so far escaped the most searching scrutiny.

Is it too much to hope that further study and research in this *problem* will bring with it the key to the essential nature of malignancy itself?

I am greatly indebted to my many friends and colleagues who have so kindly allowed me access to the notes of their cases and supplied me with valuable information.

BIBLIOGRAPHY

AHUMADA, J. C. (1943), *Tratado Elem. Ginec., Buenos Aires*, **2**, 2.
ALLEN, E. (1933), *Amer. J. Obstet. Gynec.*, **26**, 803.
ARMYTAGE, GREEN (1933), *Brit. med. J.*, **1**, 602.
ATKINSON, W. B., and LEATHEM, J. H. (1945), *Proc. Soc. exp. Biol., N.Y.*, **58**, 283.
BARNES, JOSEPHINE (1945), *Proc. R. Soc. Med.* (1945), **38**, No. 7, 324.

BARTLETT, R. W. (1935), *Amer. J. Surg.*, **29**, 123.
BELL, W. BLAIR (1922), *J. Obstet. Gynæc.*, **29**, 443.
BERKELEY, C. (1926), *J. Obstet. Gynæc.*, **33**, 657.
BLAIKLEY, J. B. (1941), *Proc. R. Soc. Med.*, **39**, 810.
BONNEY, VICTOR (1926), *J. Obstet. Gynæc.*, **33**, 658.
— — (1938), *Ibid.*, **45**, 250.
BOURNE, A., and WILLIAMS, LESLIE, *Recent Advances in Obstetrics and Gynæcology*. London: Churchill.
CLAYE, A. M. (1938), *Clin. J.*, **67**, 75.
CULLEN, T. S. (1920), *Arch. Surg.*, **1**, 215.
DOUGAL, D. (1923), *J. Obstet. Gynæc.*, **30**, 224.
— — (1938), *Amer. J. Obstet. Gynec.*, **35**, 373.
DUNCAN, C. J. (1934), *New. Engl. J. Med.*, **211**, 24.
GOUGH, W., and STEWART, C. E. (1914), *J. Obstet. Gynæc.*, **26**, 88.
HALBAN, J. (1925), *Arch. Gynæk.* **124**, 457.
HANDLEY, W. SAMPSON (1910), *Lancet*, **1**, 978.
HARBITZ, J. F. (1934), *Acta chir. Scand.*, suppl. 30.
IWANOFF, N.. S (1889), *Mschr. Œburtsh Gynæk.*, **7**, 295.
JEFFCOATE, T. N. A., (1932), *Lancet*, **1**, 662.
— —. (1934), *J. Obstet. Gynæc.*, **41**, 684.
JUDD, E. STARR (1921), *Surg. Clin. N. Amer.*, **1**, 1271.
KAULICH, L., and GOMORI (1934), G., *J. Obstet. Gynæc.*, **41**, 63.
LOCHRANE, C. D. (1923), *Ibid.*, **30**, 443.
MACLEAN, N. J. (1936), *Canad. med. Ass. J.*, **34**, 253.
MACLEOD, D. H. (1932), *Proc. R. Soc. Med.*, **25**, 138.
— — (1934), *J. Obstet. Gynæc.*, **41**, 385.
MALIPHANT, R. G. (1933), *Ibid.*, **40**, 957.
MEIGS, J. V. (1941), *Ann. Surg.*, **114**, 866.
MOIR, J. CHASSAR (1944), *Proc. R. Soc. Med.*, **37**, 437.
MORRIS, R. T. (1892), *Ann. Surg.*, **15**, 326.
MORSON, CLIFFORD (1927), *Brit. J. Surg.*, **15**, 264.
NIXON, W. C. W. (1940), *Lancet*, **1**, 405.
O'CONNOR, V. J., and GREENHILL, J. P. (1945), *Surg. Gynec. Obstet.*, **80**, 113.
PATTON, C. L., and PATTON, R. J. (1941), *Amer. J. Surg.*, **53**, 265.
PHILLIPS, MILES (1936), *Clin. J.*, **65**, 48.
PHILLIPS, R. B. (1934), *J. Obstet. Gynæc.*, **41**, 165.
REYNOLDS, L. R. (1939), *J. Urol.*, **41**, 157.
ROQUES, F. (1928), *J. Obstet. Gynæc.*, **21**, 538.
SAMPSON, J. A. (1921), *Arch. Surg.*, **3**, 245.
— — (1922, a), *Ibid.*, **5**, 217.
— — (1922, b), *Amer. J. Obstet. Gynec.*, **4**, 451.
— — (1924), *Surg. Gynec. Obstet.*, **38**, 287.
— — (1928), *Amer. J. Obstet. Gynec.*, **16**, 461.
— — (1930), *Ibid.*, **20**, 443.
— — (1940), *Ibid.*, **40**, 549.
— — (1945), *Ibid.*, **50**, 597.
SCHOFIELD, J. D., and BACON, H. E. (1938), *Ann. Surg.*, **107**, 1022.
SHAW, W. FLETCHER, and ADDIS, W. R. (1922), *J. Obstet. Gynæc.*, **29**, 452.
SCOTT, R. B. (1944), *Amer. J. Obstet. Gynec.*, **47**, 608.
THOMSON, A. P. (1926), *Proc. R. Soc. Med.*, **19**, 16.
VARTAN, C. K. (1937), *J. Obstet. Gynæc.* **44**, 715.
WEIGHTLANDT, J. A. (1934), *Proc. R. Soc. Med.*, **27**, 1493.
WHITEHOUSE, BECKWITH (1926), *Ibid.*, **19**, 16.

SOME RECENT ADVANCES IN PANCREATIC AND BILIARY TRACT SURGERY

INCLUDING SOME CASE REPORTS

BY T. M. J. D'OFFAY, LEICESTER

CARCINOMA arising from the duodenal papilla, ampulla of Vater, and the distal inch of the common bile-duct and pancreatic duct (henceforth in this paper called ampulla-papillary carcinoma) must be carefully distinguished from carcinoma of the head of the pancreas proper, because although they have many features in common, they differ fundamentally in their pathology and prognosis, and to a lesser degree in their clinical course. But owing to the difficulty of diagnosing one from the other both before and at operation, and because of the similarity of the surgical procedure employed for both, these two lesions are best considered together—due emphasis being paid to their dissimilar features. The ampulla-papillary group of carcinomas are considered as of one type because of the impossibility of determining the exact site of origin in many cases, even after careful microscopical examination (Ewing, 1928; Hunt and Budd, 1935; Outerbridge, 1913).

Carcinomas in this region have hitherto been looked upon as the most formidable of diseases, and have shared the honours with carcinomas of the œsophagus as the last to yield to the skill and ingenuity of the surgeon, who has been greatly helped in this respect during the last 10 years by advances in biochemistry and in blood and plasma transfusions. The age of the patient, the deep retroperitoneal position of these growths, their close relationship to vital blood-vessels, and the development of jaundice with subsequent failure of the liver functions are the factors which gave a practically hopeless outlook to these lesions—so much so that in 1905 Lord Moynihan, when discussing the few cases (13 in number) that had been subjected to operation up to that time, wrote: "The results serve to show that the mechanical difficulties of the operation are well-nigh insuperable, and that if boldness and good fortune are the operator's gifts, the result to the patient hardly justifies the means". This attitude prevailed and was reiterated by surgeons from time to time until 1935, when Whipple, Parsons, and Mullins (1935) stimulated new interest in this subject by their able advocacy of a new two-stage operation.

AETIOLOGY AND INCIDENCE

Nothing is known of the aetiology of the disease.

Carcinoma of the ampulla-papillary region occurs in 1·13 per cent of all carcinomas, and 0·09 per cent of all autopsies (Lieber, Stewart, and Lund, 1939). It is an uncommon tumour, 222 verified cases having been reported in the literature up to 1939 (Lieber et al., 1939). The age and sex incidence is the same as for carcinoma of the pancreas.

Carcinoma of the pancreas accounts for 3 per cent of all cancer deaths (Clute, 1936). The age incidence is similar to that of carcinoma of the stomach, and the male/female ratio is 2 to 1. Rives,

Romano, and Sandifer (1937) give the following ratio of distribution from a collective review of 415 cases: head 66 per cent, body and head 18 per cent, body 6 per cent, and tail 10 per cent (*Fig.* 154).

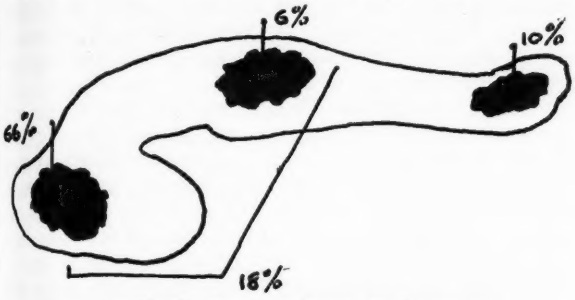


FIG. 154.—Distribution of carcinomata in the pancreas. (*After Rives, Romano, and Sandifer*, 1937.)

The relative incidence of ampulla-papillary carcinoma and carcinoma of the head of pancreas has received scant attention. Accuracy demands confirmation of the diagnosis by biopsy or autopsy. The figures given by Schnedorf and Orr (1941) and by Ransom (1938) alone meet these requirements, but are perhaps too small to be of value. The proportion of carcinoma of head of pancreas to ampulla-papillary carcinoma is approximately 8 to 5.

PATHOLOGY

Ampulla-papillary Carcinoma.—All pathologists who have studied the subject are agreed that carcinomas arising from the ampulla-papillary region are of two types: a papillomatous growth projecting into the duodenum and measuring ½–3 in. in diameter; or a flat ulcerating growth which may be so small as to remain undetected in a hurried examination or simulate a stricture of the lower end of the common bile-duct. These tumours remain localized for a relatively long time, rarely invading the head of the pancreas, and are late in metastasizing to the regional lymph-nodes. Such favourable features are probably due to the early onset of jaundice, and were first pointed out by Outerbridge (1913), who found metastases in 22 per cent of 110 cases reported in the literature up to that time. Dick (1939) has recently confirmed those findings. A review of the reported cases (*Table I*) reveals that lymph-node metastases occurred in 24·6 per cent of the cases.

Table I.—INCIDENCE OF LYMPH-NODE METASTASES IN AMPULLA-PAPILLARY CARCINOMA

Author	*No. of Cases*	*Metastasis Present*
Perry and Shaw (1893)	15	3 (20 per cent)
Outerbridge (1913)	110	24 (22 per cent)
Cohen and Colp (1927)	4	0
Whipple et al. (1935)	7	2 (28 per cent)
Ransom (1938)	5	0
Bagenstoss (1938)	28	15 (53·6 per cent)
Dick (1939)	13	1 (7·7 per cent)
	182	45 (24 per cent)

Carcinoma of the Head of Pancreas.—The head alone is the seat of growth in 40–66 per cent of cases. The growth arises from the epithelium of the acini or smaller ducts, and is usually hard and scirrhous. The grade of malignancy is unfortunately high in most cases (81 per cent), corresponding to gastric carcinoma in this respect. Drapiewski (1944), of the Mayo Clinic, found the following grades of malignancy in 82 carcinomas of the pancreas: Grade 1, 1 case (1·2 per cent); Grade 2, 14 cases (17 per cent); Grade 3, 33 cases (40 per cent); Grade 4, 34 cases (41 per cent). "Because of its highly infiltrative growth, the exact limits of the neoplasm are not discernible. Indeed, microscopical section usually shows extension of malignant cells at some distance into pancreatic tissue which appears grossly uninvolved." (Brunschwig, 1942) (*see Case* 3 reported here). At operation or autopsy invasion of the duodenal wall, mesocolon, and adjacent blood-vessels is a frequent finding. Finally, metastases to lymph-nodes, liver, and lungs occur relatively early. Drapiewski (1944) found metastases in lymph-nodes in 73·2 per cent of 82 cases examined. All reports agree that early and extensive metastases are the rule, and that the disease is rapidly fatal, the illness averaging 4½ months. Of 12 cases reviewed at autopsy, Ransom (1938) found only 2 cases operable, one in the head, the other in the body. Of 46 cases of carcinoma of the head of the pancreas and ampulla-papillary region seen during a 2-year period (1942–4), Cattell (1945) reports 3 radical excisions for carcinoma of the head of the pancreas, whereas there were 13 such excisions for ampulla-papillary carcinoma.

DIAGNOSIS

Clinical Features.—Carcinoma of the ampulla-papillary region and to a less extent of the head of the pancreas possesses only one good feature: the development of jaundice at a relatively early stage of the disease. Classically the jaundice is painless and progressive; but recent studies have definitely proved that in a high proportion of cases the jaundice is neither painless nor progressive.

Icterus occurs in practically all cases of carcinoma of ampulla-papillary region. It was present in 105 of 110 cases reported by Outerbridge (1913), and in all 52 cases reviewed by Pallin (1920). In carcinoma of the head of the pancreas, jaundice was observed in 86 per cent (Rives et al., 1937) and 81 per cent of cases (Berk, 1941), and severe abdominal pain in 46 per cent of cases (Drapiewski, 1944). When carcinoma of all parts of the pancreas are considered the incidence of jaundice is naturally lower (71 per cent) and pain higher (76 per cent) (Drapiewski, 1944). Indeed, pain is a major complaint in such cases. In Ransom's series (1938) of 109 cases of carcinoma of the pancreas and bile-ducts, abdominal pain was the major complaint in 45 per cent of cases and the *most common initial complaint*. In half the cases the pain was felt in the epigastric region and had a continuous boring character, going through to the back. In the others the pain closely resembled mild attacks of biliary colic. Pain associated with carcinoma of ampulla-papillary region is almost invariably of the latter type.

Loss of weight, anorexia, and asthenia are early, rapidly progressive features in carcinoma of the head of the pancreas, usually preceding the jaundice; whereas in ampulla-papillary carcinoma they are less marked and manifest themselves only after the onset of jaundice (Cattell, 1945).

Reflex vomiting occurs in one-third of the cases, while progressive vomiting due to duodenal obstruction by growth occurs occasionally as a late or the only presenting feature of the disease. Severe constipation is present in over a third of cases (36 per cent) and steatorrhœa in 12 per cent (Ransom, 1938).

Fever and rigors occur occasionally, and may be conspicuous features (*Case* 4, herein described).

Examination of the abdomen usually reveals the edge of an enlarged liver, and may reveal the presence of a pancreatic tumour (23 of 35, i.e., 65 per cent, cases of carcinoma of pancreas—Schnedorf and Orr, 1941) (tumours of the ampulla-papillary region are usually too small to be palpable), secondaries in the liver and peritoneum, free fluid in the peritoneal cavity, and finally a dilated gall-bladder. One should be chary of concluding inoperability when there is free fluid in the peritoneal cavity. The writer has observed two cases of carcinoma (one pancreatic, one gastric) during 1945, in which free fluid in the peritoneal cavity was caused by starvation hypoproteinæmia. In both cases the free fluid completely disappeared after the administration of a high protein and carbohydrate diet.

Dilatation of the gall-bladder is present in nearly all cases. Its detection clinically or at peritoneoscopy or operation is of such diagnostic importance that we should pause to consider its significance in some detail. Courvoisier's law, which is concerned with the problem, states literally: "With obstruction of the common duct by stone, dilatation of the gall-bladder is rare; the organ being usually shrunken. With other types of obstruction, on the other hand, dilatation is the rule; shrinking occurring in only one-twelfth of the cases". This law was enunciated by L. G. Courvoisier in a small monograph dealing with the pathology and surgery of the biliary tract published in 1890. In this he described the condition of the gall-bladder in 187 cases of obstruction of the common bile-duct with jaundice. In 87 cases the obstruction was due to stones, and of these 80 per cent had a shrunken gall-bladder. On the other hand, in 92 per cent of the 100 cases, in which obstruction was due to causes other than stones, the gall-bladder was distended. Courvoisier went on to explain this striking difference by pointing out that infection of the gall-bladder wall, nearly always present with stones, produced so much fibrosis that dilatation was impossible. With other causes the thin-walled bladder was able to dilate, sometimes to large proportions.

This law has since been fully confirmed repeatedly: Cabot (1909) gives the following figures for 86 cases studied: 57 cases of stones in common bile-duct—in 2 only was the gall-bladder dilated; whereas in 29 cases of carcinoma of the head of the pancreas, only 2 did not present a distended bladder. Thus 4 cases out of 86 proved the exception to the rule. Ransom (1938) found a dilated gall-bladder in 83 per cent of 109 cases of obstructive jaundice due to carcinoma.

Clinically it is possible to feel the distended gall-bladder in only half the cases (Eustermann, 1922, 138 cases studied). Thus Courvoisier's law is of much greater practical importance at peritoneoscopy or laparotomy than at the bedside.

Radiography.—In Lieber, Stewart, and Lund's (1939) series positive evidence of a lesion in the region of the papilla was found in 16 of 60 cases (27 per cent). In carcinoma of the head of the pancreas positive evidence in the form of widening of the duodenal curve or compression or invasion of the duodenal lumen usually signifies a late stage of the disease (Brunschwig, 1942).

Tests of Liver Function.—These tests are most useful and constitute an important advance in the diagnosis and treatment of jaundiced states. It is well to know that these tests have certain limitations, and to remember that the liver is an organ performing many functions, which are not equally injured by a disease process or toxic agent. There is "dissociation of the functional activities of the liver" (Mann, 1942). The value of the tests is that, on the one hand, they reliably assist in the diagnosis between obstructive and hepatogenous jaundice, and on the other they detect the presence or absence of liver insufficiency. A knowledge of the latter is, of course, of the greatest importance in guiding the pre-operative treatment, the type of operation and post-operative management, and in the prognosis.

The absence of bile in the duodenal contents and fæces, and the absence of urobilinogen from the urine signify complete obstruction of the bile-ducts—the classical features of obstructive jaundice, in the early stages of which the liver functions are normal. On the other hand, there is the typical picture of hepatogenous jaundice with bile in the stools and increased amount of urobilinogen in the urine with failure of the liver functions early in the disease. Now, if these two types of jaundiced states remained as such, or if all cases of jaundice could be seen within one or two weeks of onset, there would be little difficulty in distinguishing one from the other. But the types tend to merge: obstruction is complicated by hepatitis; and hepatitis by obstruction. In obstructive jaundice due to stone or neoplasm in the bile-passages, liver damage is brought about by continued back pressure or by infection or both. In pure hepatogenous jaundice (toxic, infective, and cirrhotic), intrahepatic biliary obstruction occurs in 20 per cent of cases (Steigmann and Popper, 1943). Indeed, these authors found evidence of some obstruction in nearly all cases of hepatitis; but complete obstruction of 2–30 days' duration occurred in 20 per cent of 127 cases studied. The liver in these cases shows dilatation of the small bile capillaries within the parenchyma all the way to the portal triads (Popper, 1936; Hanger, 1940). According to Aschoff (1937) "The morphologic picture localizes the obstruction in the region of the periportal fields at the junction between the bile capillaries and bile-ducts. Here the most vulnerable part of the biliary system is localized".

Steigmann and Popper (1943) proceed to show that the simplest way of recognizing the real nature of these cases of hepatitis complicated by intrahepatic obstruction is to determine the urobilinogen excretion in the urine for a prolonged period. An isolated estimation may give an erroneous impression. "A continuous observation of the urobilinogen excretion throughout the course of the disease would

show first an increase, then a decrease (period of obstruction), and then again a rise before improvement, and finally a drop to normal levels."

Urinary urobilinogen originates from changed bilirubin, which is absorbed from the intestinal tract. Normally the liver oxidizes nearly all the urobilinogen re-absorbed from the intestine; and small amounts only appear in the urine (up to 3 mg. in 24 hours). Liver damage interferes with this oxidation; and large amounts of urobilinogen are therefore excreted daily in the urine (5–300 mg.), the amount depending on the severity of the liver damage. Complete obstructive jaundice, on the other hand, shows a gradual but persistent decline in traces of urobilinogen found initially in the urine. A small amount of urobilinogen (up to 5 mg.) may be present in the fæces, this being derived from bile-stained débris coming from the wall of the intestine (Steigmann and Dyniewicz, 1943).

The plasma prothrombin response to intravenous injection of vitamin K is another test used in the differential diagnosis of jaundice. The normal prothrombin content of plasma is maintained by adequate absorption of vitamin K from the bowel, and its conversion into prothrombin by the liver. And prothrombin deficiency may be due to the failure of either or both these functions. In the absence of intestinal bile, the resulting prothrombin deficiency is rapidly corrected by giving vitamin K (a quick response). Conversely, prothrombin deficiency due to hepatitis cannot be, or is slowly, corrected by administering vitamin K (no response, or a slow response). Olwin (1941), Lord and Andrus (1941), Allen and Julian (1942), and Allen (1944) are all agreed that this test offers an accurate method of differential diagnosis in early cases of jaundice and in the absence of infection complicating obstruction. They emphasize the importance of observing certain technical details in performing the test. This test is also a valuable one for estimating hepatic insufficiency—" the most practical we now have " Ivy and Roth (1943).

In some respects all tests of hepatic functions are helpful in the differential diagnosis of jaundice, provided they are done in the early stages of jaundice; that is, before the types tend to merge. They indicate poor function in cases of hepatitis, and give normal results in early cases of pure obstruction. The galactose tolerance test of Bauer (1906), hippuric acid synthesis test of Quick (1932), and cephalin-cholesterol flocculation test of Hanger (1939) have all proved useful in this respect; but their chief merit lies in determining damage and residual function (Ivy and Roth, 1943). The bromsulphthalein (5 mg.) clearance test of Rosenthal (1922) is unreliable in the presence of jaundice, but one of the most sensitive indices of liver damage in the absence of jaundice.

Aspiration Liver Biopsy has proved of great value in the diagnosis of obscure and difficult cases. The technique has been well described by Sherlock (1945). It has definite limitations and is not without danger—0·67 per cent deaths from bleeding (Sherlock). Failure to aspirate a cylinder of tissue occurred in from 2–10 per cent of Sherlock's cases. The specimen having been obtained, however, there is no difficulty in coming to a diagnosis, except in old-standing cases of biliary obstruction, which give a picture not unlike an ordinary cirrhosis.

Peritoneoscopy.—This procedure is, in my estimation, of much greater value than aspiration biopsy, since it allows one to view the liver, gall-bladder, and the peritoneal cavity generally; and to take a biopsy of the liver. This is done with less risk (0·2 per cent mortality, Ruddock, 1937) than aspiration liver biopsy, and with as little discomfort to the patient. I have used this method of investigation and diagnosis on several occasions and found it most useful, if only to prove or disprove the presence of secondary growths. Biopsy of the liver is easily and safely carried out under vision, and hæmostasis secured by electro-coagulation.

Diagnosis at Operation.—There is usually on difficulty in coming to a diagnosis in those carcinomas of the ampulla-papillary region which project into the duodenal lumen. But those which invade the pancreas and those arising from the head of the pancreas proper may give rise to acute diagnostic problems. Chronic pancreatitis localized to the head (usually), and adenoma (rarely), and calcification of the pancreas (6 recorded cases—Gordon Taylor, 1942; Whipple, 1945) may so closely simulate carcinoma as to be indistinguishable from it. Almost every experienced abdominal surgeon has at some time seen a case of chronic pancreatitis mistaken for carcinoma and vice versa (*see Case* 5). Walters and Dehne (1932) estimated that 15 per cent of cases diagnosed as carcinoma of the pancreas were inflammatory lesions as judged by the end-results of short-circuit operations. Sir John Fraser (1938), in his survey of 1035 cases of jaundice treated between 1927 and 1937, stated that " 7 per cent of simple cases are mistaken for malignant disease, and 16 per cent of malignant cases are classified as simple ". Cattell (1945) reports a mistaken diagnosis in at least 5 (10 per cent) of 51 cases diagnosed as carcinoma of the pancreatico-duodenal region during a two-year period. Four of these had a preliminary cholecyst-jejunostomy performed; later at the second operation nothing was found in three, and a simple tumour in the fourth. The fifth case was subjected to a successful resection of the duodenum and head of the pancreas, but the lesion proved to be pancreatitis.

Examinations of frozen sections " while you wait " at operation may prove either misleading owing to pancreatitis accompanying the tumour and overlying carcinoma tissue (Brunschwig, 1942) or useless (Cattell, 1945). Moreover, there are cogent objections to incising a pancreatic tumour unless one is prepared to carry out an immediate resection. It would seem that the best procedure to adopt in cases of doubt is to do a preliminary cholecyst-jejunostomy; and if enlarged lymph-nodes are present to remove one or more for microscopical examination. The second operation, if indicated after lymph-node biopsy, will in most cases reveal the true diagnosis, as in Cattell's cases.

TREATMENT

Prior to 1937, when Brunschwig performed successfully a modified Whipple operation for carcinoma of the head of the pancreas, few attempts had been made at the radical removal of this growth.

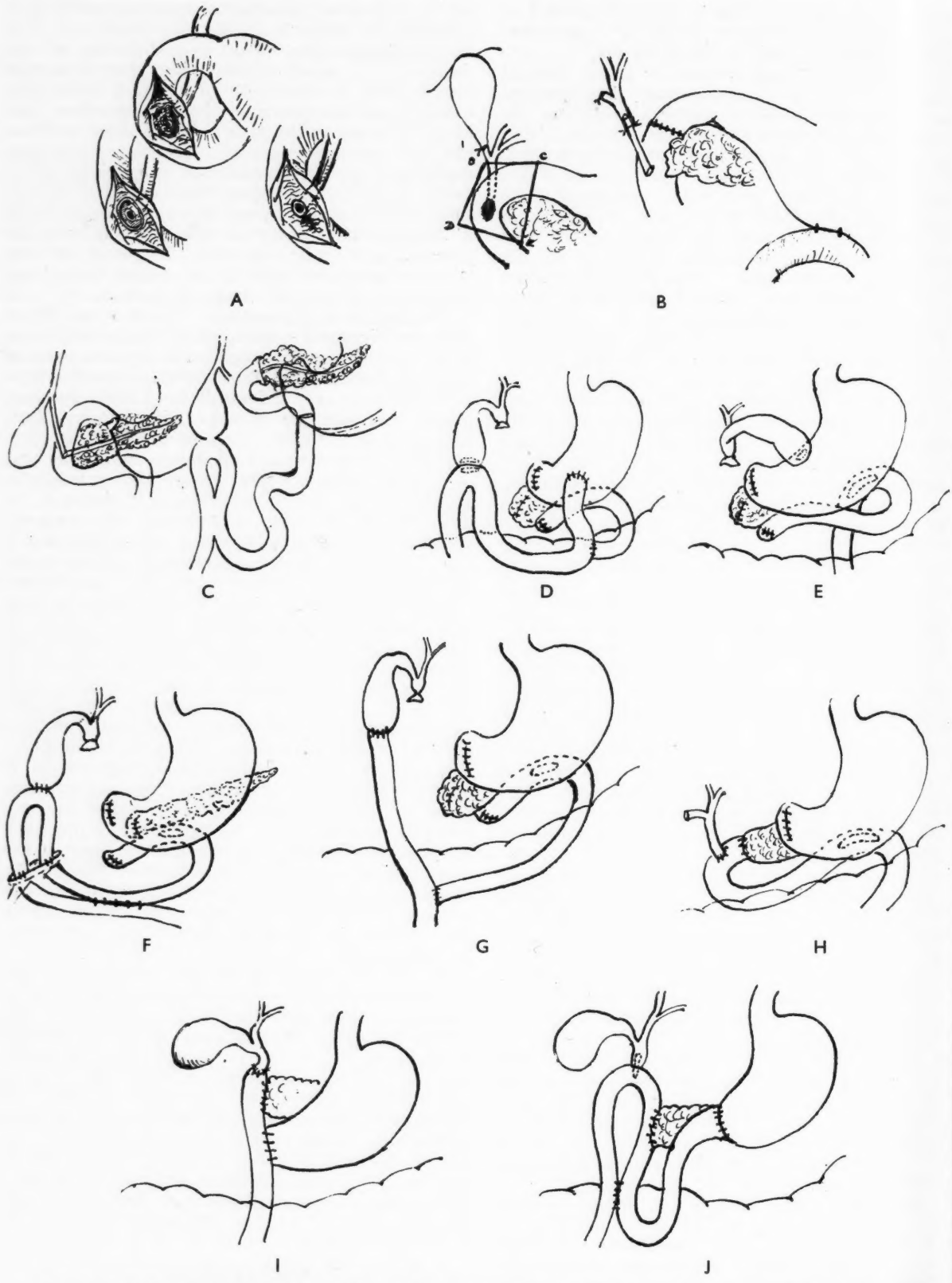


FIG. 155.—*Early operations for ampulla-papillary carcinoma*: A, Simple transduodenal excision; B, Hirschel's (1914) operation, an example of limited cylindrical excision of the duodenum with wedge excision of head of pancreas and end-to-end anastomosis of duodenum; C, Kausch's operation (1912) (two-stage) for radical excision of head of pancreas and duodenum—*first operation for carcinoma of head of pancreas*: D, Codivilla's radical pancreatico-duodenectomy performed in 1898—*radical operations carried out since* 1935 *for both types of cancer*: E, Whipple, Parsons, and Mullins (1935); F, Brunschwig (1937); G, Whipple (1938); H, Hunt (1941); I, Whipple (one-stage) (1943); and J, Cattell (1943).

Codivilla in 1898 resected the duodenum and head of pancreas in one stage and by a technique which is very similar to those now in vogue (*Fig.* 155, D). To him, therefore, should priority be accorded in the development of radical pancreatico-duodenal resection. His patient died 24 days after operation, Tuffier (1905) and Michaux (1906) also carried out resection of the duodenum and head of the pancreas, but without success.

Carcinoma of the ampulla-papillary region, on the other hand, by virtue of its intraduodenal position, its small size, and very localized nature, has proved more amenable to surgical extirpation. In 1898 Halsted (1899) performed the first excision of such a cancer. The operation consisted of a wedge resection of duodenum and growth, and the patient died from a local recurrence nine months later. From then on until 1935, when Whipple, Parsons, and Mullins described their two-stage radical operation for this lesion, no less than 78 reports of limited excision of these tumours appeared in the literature. These early operations were of two types: in most cases a simple and crude removal of the tumour was carried out after gaining access by opening the anterior wall of the duodenum (*Fig.* 155, A); in a few cases (5 reports, including Halsted's original case) (Mayo-Robson, 1900; Korte, 1904; Hirschel, 1914; Tenani, 1922), the operation consisted of a cylindrical or wedge resection of the duodenum, and wedge removal of the adjacent portion of the pancreatic head containing the tumour (*Fig.* 155, B). Kausch in 1912 devised and performed a two-stage radical excision of the duodenum and head of the pancreas, which was in many respects similar to the present-day operation (*Fig.* 155, C). His patient died nine months later from acute cholangitis without evidence of metastases.

The operative mortality for these operations was 29 per cent for transduodenal excision, and 45 per cent for cylindrical duodenal excision (Hunt, 1941). It is interesting to compare these figures with the operative mortality-rate following simple laparotomy (36 per cent), cholecyst-gastrostomy and cholecyst-jejunostomy (32 per cent), and simple cholecystostomy (70 per cent) (Cohen and Colp, 1927). Sir John Fraser (1938) in his review gave the following mortality figures for simple internal short-circuit operations: 40 per cent for all cases of obstructive jaundice; 51 per cent in malignant disease of the pancreas, and 19 per cent in chronic pancreatitis. In more than half the cases (58 per cent), the cause of death was cholæmia; shock, peritonitis, and hæmorrhage in equal proportion accounting for the other deaths. He also pointed out the close relationship that exists between the degree of jaundice and the mortality-rate (the deeper the jaundice, the higher the mortality). With regard to the favourable mortality figures quoted above for transduodenal and other excisions as compared with cholecyst-gastrostomy, it must be remembered that unsuccessful cases are seldom reported. If these, then, are taken into consideration, the mortality percentage for excision may well have equalled or even surpassed that following cholecyst-gastrostomy.

Thus the position stood until 1935: three unsuccessful attempts at radical removal of the duodenum and head of the pancreas for carcinoma of head of the pancreas, and two successful two-stage limited radical resections for carcinoma of the ampulla-papillary region by Kausch (1912) and Tenani (1922). The other successful excisions, with the exception of Hirschel's, had been of a very limited character, and performed through an opening in the duodenum.

As a result of certain mistakes and a fatality in 1934, Whipple, Parsons, and Mullins (1935) re-examined this problem with a view to eliminating the defects of the previous operations and devising a safer and more radical procedure. They pointed out the error of performing the operation in one stage; and they recognized that the resection as hitherto carried out had been limited in extent in order to preserve as much duodenum as possible, and to safeguard the entrance of pancreatic juices into the intestine, both of which were generally considered essential for life. Whipple and his colleagues, however, knew that the dog could survive total duodenectomy, a demonstration of the greatest importance which was first made by Dragstedt et al. in 1918 and later confirmed by Moorhead and Landes (1919) and Mann and Kawamura (1919). Fortified by this knowledge, and by the fact that patients suffering from carcinoma of papilla of Vater are able to survive for months without pancreatic external secretions (Whipple, 1941), Whipple, Parsons, and Mullins were prepared to sacrifice both the duodenum and pancreatic juices in order to plan and carry out a truly radical resection for carcinoma. This they did successfully for ampulla-papillary carcinoma in 1935. The operation was performed in two stages; the first stage consisting of cholecyst-gastrostomy and gastro-jejunostomy; resection of most of the duodenum and of head of the pancreas with closure of the duodenal ends and pancreatic stump was performed two weeks later (*Fig.* 155, E). The patient made a rapid recovery; and there was no subsequent interference with the digestion of fat and protein, or with carbohydrate metabolism. This operation represents a milestone of progress in the surgery of the pancreas, duodenum, and biliary tract; and with some modifications it has now been carried out on at least 125 occasions since 1935.

Brunschwig in 1937, in carrying out the first successful excision of carcinoma of the head of the pancreas by this technique, modified the operation in some details (*Fig.* 155, F); modifications which have since become standard procedures. He anastomosed the jejunum instead of the stomach to the gall-bladder, with an accompanying jejunojejunostomy. This was intended to minimize or prevent the occurrence of cholangitis, which had been such a frequent complication of cholecyst-gastrostomy. Moreover, cholecyst-jejunostomy was much less of a technical handicap at the second operation than cholecyst-gastrostomy. Brunschwig further extended the resection in all directions. He removed the whole duodenum and the whole head of the pancreas, sectioning the latter through the neck.

At about the same time a new and powerful impetus was given to this branch of surgery by the discovery of vitamin K by Dam (1935), by Quick's (1935) observation that prothrombin deficiency was the cause of bleeding, and by the realization that the

latter could be remedied by the former. This, coupled with a better understanding of shock and its prevention and treatment by blood and plasma transfusions, have contributed immensely in transforming a formidable and dangerous and consequently rare undertaking into a relatively safe and common one. Thus, in 1940, it became possible to carry out successfully radical pancreatico-duodenectomy in one stage: Whipple (1945) for carcinoma of the head of the pancreas, and Hunt (1940) for ampulla-papillary carcinoma (*Fig.* 155, H). By April, 1942, at least 64 radical pancreatico-duodenectomies had been performed; and the time had come for a critical review of the results achieved. The main interest was now focused on the harmful effects of pancreatic exclusion. The fistula, the common bile-duct should be anastomosed to the jejunum in one-stage resection, and its cut end should be tied and inverted with silk sutures or anastomosed to the jejunum in staged operations in which a cholecyst-jejunostomy is performed first.

At the time of writing no less than 125 cases of radical pancreatico-duodenectomy have been reported since 1935 (Orr, 1945, 104 cases; Cattell, 1945, 18 cases (5 included in Orr's report); Cole and Reynolds, 1945, 5 cases; and 3 personal cases reported here). The operative mortality has been 28 per cent. The best series of cases reported so far is that of Cattell (1945) who, during a two-year period (August, 1942–August, 1944), performed 17 radical pancreatico-duodenectomies (12 for carcinoma

Table II.—Operative Mortality

		No. of Cases	*Deaths*	*Percentage*
Transduodenal excisions (Hunt, 1941):—				
1898–1925		46	20	45
1925–1941		47	7	15
Radical pancreatico-duodenectomy:—				
1935–1942 (Whipple, 1942)	Two-stage	41	12	29·2
	One-stage	23	8	34·7
1942–1944 (Orr, 1945)	Two-stage	15	4	26·6
	One-stage	19	6	31·5
1942–1944 (Cattell, 1945)		17	2	11·8

Table III.—Results of Palliative Biliary Entero-anastomosis

Author	*No. of Cases Operated On*	*Operative Mortality*	*No. of Cases Followed*	*Average Length of Life after Operation* (mths.)
Judd and Parker (1928)	34	47·0	12	7·7
Coller and Winfield (1934)	30	26·6	19	7·2
Fraser, Sir J. (1938)	211	51·84	140	*
Sallick and Garlock (1942)	50	48·0	25	6·3
Gray and Sharpe (1943)	11†	63·6	4	15·5

* 66 per cent dead in less than six months, and the rest within a year; but during life a large percentage enjoyed freedom from pain and jaundice.
† All carcinomas of ampulla-papillary region only.

Table IV.—Results of Local Excision of Ampulla-papillary Carcinoma (*After Hunt*, 1941)

No. of Cases Operated On	*Operative Mortality Per cent*	*No. of Cases Followed*	*Average Length of Life of Patient Dead when Reported*	*Average Length of Life of Patients Living when Reported*
109	31·1	56	17·4 months (24 cases)	32·5 months (32 cases)

mortality of the operation had been 32 per cent (Whipple, 1942); and according to Cattell (1943) the commonest cause of death had been the development of internal pancreatic fistulæ. A small number of deaths had been caused by bile peritonitis due to sloughing of the ligated end of the common bile-duct. Of 41 cases of radical resection reported by Whipple (1941), 8 developed an external pancreatic fistula and 5 a biliary fistula. The former closed spontaneously, but the latter often did not. Moreover, a proportion of patients developed some impairment of carbohydrate metabolism as shown by varying degrees of diabetes; and others, probably a third, had deficient absorption of fat and protein.

Owing to these complications, Whipple (1943) (*Fig.* 155, I) and Cattell (1943) (*Fig.* 155, J), decided to re-establish the flow of pancreatic juices into the intestine by anastomosis of the pancreatic stump, or duct if dilated, to the jejunum or stomach; and this has now become a routine procedure wherever possible. To prevent bile peritonitis and bile

of ampulla-papillary region, 3 for carcinoma of head of pancreas, 1 for carcinoma of duodenum, and 1 no carcinoma found) with two operation deaths (11·8 per cent) (1 dying of shock and anuria 24 hours after operation, and the other of pyelonephritis and pericarditis two months after operation). In all these the pancreatic stump was anastomosed to the jejunum; and resection was performed in two stages in 13, and in one stage in 4.

PROGNOSIS

The immediate mortality figures for the various types of operation are given in *Table II.* It is noteworthy that Cattell's personal cases, in most of which the operation was performed in two stages, and included anastomosis of the pancreatic stump to the jejunum, should show such a low mortality. It is a most encouraging picture. There are as yet no comparative operation mortality figures for carcinoma of ampulla-papillary region and for carcinoma of pancreas.

The late results are known for palliative operations (*Table III*) and for localized transduodenal excision (*Table IV*), but it is as yet too early to judge the late results of the radical operation. When comparative studies are made, however, it will be necessary to take into account the fact that radical operations have been performed for both carcinoma of the head of the pancreas and carcinoma of ampulla-papillary region (Whipple, 1941), whereas the limited transduodenal excision was performed only for the latter group of carcinoma, the malignancy of which differs radically from that of the former. The localized nature and low malignancy of these ampulla-papillary growths is reflected in the long survival of so many cases following such a limited operation. GordonTaylor (1942) and Brunschwig (1942) report 10 cases that lived over 5 years and 4 over 4 years.

CASE REPORTS

Case 1.—**Papillomatous adenocarcinoma of ampulla-papillary region treated by simple transduodenal excision.**

Mrs. A. M., aged 71 years, admitted on March 3, 1943. First attack of painless jaundice 11 months before, which lasted 10 weeks; it was accompanied by some loss of weight, and then cleared up completely. Recurrence of painless jaundice two months before admission with pale stools and bile in urine. Slight loss of weight; but patient had gained some weight following the first attack of jaundice.

On Examination.—A thin and tired and deeply jaundiced woman. Liver edge palpable 1 in. below costal margin. Gall-bladder dilated and palpable. Serum bilirubin, 6 mg. per cent. R.B.C., 3,510,000 per c.mm. Hb, 62 per cent. Prothrombin index, 73 per cent.

Vitamin K and blood transfusions given before operation.

Operation (March 12).—*Transduodenal excision.* The liver was enlarged and bile-stained. The gall-bladder and bile-ducts were grossly dilated and contained 'white bile'. The second part of the duodenum contained a papillomatous-like tumour. The duodenum was mobilized and opened over the tumour, which was soft and pink. It was the size of a golf ball and arose by a narrow pedicle from the papilla of Vater. There was no induration around the pedicle, or other indication of malignancy. The tumour was excised by cutting through the tissues around the base of the pedicle. The papilla of Vater was thus excised with the tumour; a small depression being left, in which the cut ends of the common bile-duct and pancreatic duct were visible. These were stitched to the margins of the defect in the posterior duodenal wall (*see Fig.* 156). The anterior duodenal wall was closed and the common bile-duct drained by an indwelling tube put into its supraduodenal portion; and the operation completed.

Pathology.—The tumour was a soft, fleshy papilloma 1¼ in. in diameter, with a short narrow pedicle surrounded by a narrow rim of duodenal tissue (*Fig.* 156). *Histologically* it was a malignant adenocarcinoma, probably arising from the duodenal papilla. There was no invasion of the excised duodenal tissue.

Progress.—The patient made a rapid recovery; jaundice cleared; she gained weight and strength, and remained in good health for two years and six months. In November, 1945, she began to lose weight; a hard mass developed in her epigastrium and increased in size. No jaundice. Laparotomy in January, 1946, confirmed the presence of a local recurrence in the head of the pancreas which was irremovable owing to invasion of the superior and inferior mesenteric veins. No secondaries were found. A posterior gastrojejunostomy and cholecyst-jejunostomy was performed. The patient died the next day (Jan. 15) from shock (?), two years and 10 months after local excision of tumour. Autopsy was not performed.

Comment.—This was clearly a carcinoma arising from the duodenal papilla that had all the appearances of a simple papillomatous tumour, and thus induced one to perform a simple local excision. A radical pancreatico-duodenectomy should have been performed at the outset, or later, when the result of the histological examination

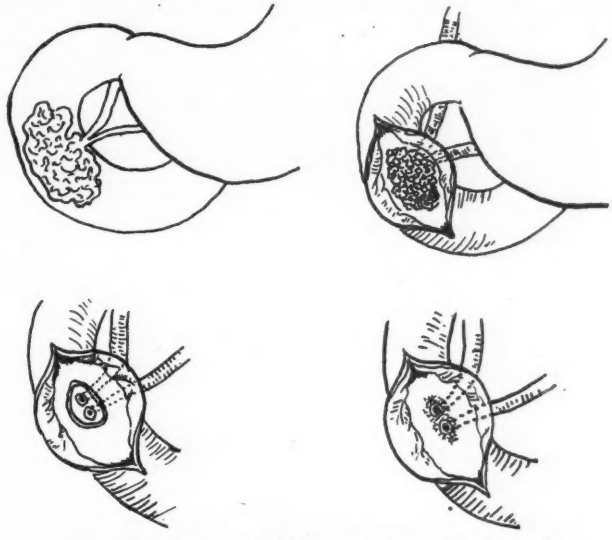


Fig. 156.—*Case* 1. Papillomatous adenocarcinoma arising from duodenal papilla removed by simple transduodenal excision, with local re-implantation of the ducts.

became known. It is noteworthy that the recurrence was purely local and might have been removable if the patient had presented herself earlier. Other interesting features in this case were the two attacks of jaundice separated by an interval of 7 months, during which the patient was perfectly well and even gained weight.

Case 2.—**Adenocarcinoma of the head of pancreas treated by one-stage radical pancreatico-duodenectomy.**

Mr. J. J. W., 70 years, admitted April 4, 1945. Pain in right hypochondrium for one month: jaundice, 15 days; pale stools and dark urine, 7 days; some loss of weight.

On Examination.—A thin and slightly wasted man, with slight jaundice. Liver not palpated; gall-bladder distended and palpable. R.B.C.'s 4,300,000; Hb 78 per cent. Serum-bilirubin 4 mg. per cent. Prothrombin index 80 per cent. Hippuric acid test, normal.

A provisional diagnosis of carcinoma of pancreatic head was made on the clinical finding of dilated gall-bladder.

Operation (April 24), under spinal anæsthesia.—*One-stage radical pancreatico-duodenectomy, with pancreatic exclusion.* Hard mass localized to head of pancreas, with distended common bile-duct and gall-bladder. Slight enlargement of the liver. No secondaries detected. The head of pancreas and duodenum were easily mobilized. There was no invasion of the portal or the superior mesenteric vein. The stomach was divided across the pyloric antrum, the pancreas through the neck, and the jejunum about 3 in. from the duodeno-jejunal flexure. The mass consisting of the whole duodenum and head of pancreas was then removed, after final separation from the portal vein and superior mesenteric vessels. The pancreatic duct was ligated with silk, and the stump closed with interrupted silk sutures. The cut ends of the stomach and jejunum were

then anastomosed; and the cut end of the common bile-duct implanted into the side of the jejunum 12 in. distant from the gastrojejunal anastomosis (*Fig.* 157). A drain was placed down to the pancreatic stump; and the abdomen closed.

PATHOLOGY (*Fig.* 158).—" A whole section to include the wall of the duodenum and the whole portion of excised pancreas shows a fairly well-differentiated adenocarcinoma of the pancreas of the scirrhous type. The uninvaded portion of the pancreas shows a good deal of cirrhotic change. The growth has invaded the mucosa of the duodenal wall beyond the muscularis mucosæ. No lymph-nodes were available for section."

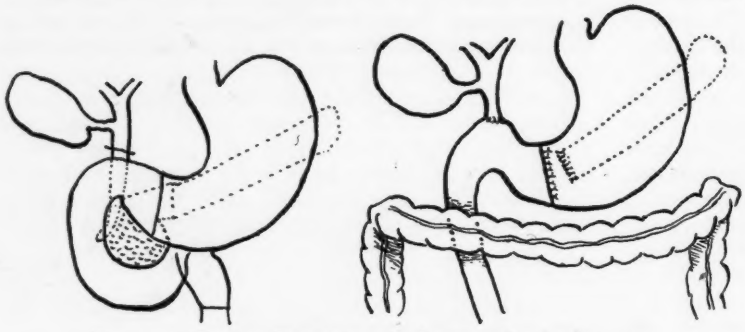


FIG. 157.—*Case* 2. One-stage radical pancreatico-duodenectomy with pancreatic exclusion.

PROGRESS.—The immediate post-operative stage passed without incident. The abdomen healed by primary union. Jaundice cleared; the patient gained weight and left hospital three weeks after operation. Carbohydrate metabolism and fat absorption were not disturbed. Six months after operation, the patient developed acute pyelitis. From then on his general condition deteriorated; secondary nodules appeared in the abdominal wall and he died 9 months after his operation from carcinomatosis. A post-mortem examination was not performed.

Comment.—A case of scirrhous adenocarcinoma of the head of the pancreas successfully removed by radical pancreatico-duodenectomy with pancreatic exclusion in one stage. There was satisfactory digestion and absorption of fat and protein. Death 9 months later from carcinomatosis.

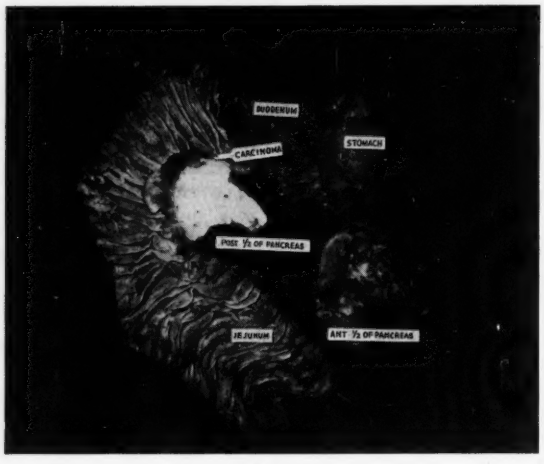
Case 3.—**Adenocarcinoma of the head of pancreas treated by radical pancreatico-duodenectomy (One-stage) with pancreatico-jejunostomy.**

Mr. D. W., aged 52, admitted July 18, 1945. Severe diarrhœa lasting a fortnight 7 weeks before admission; jaundice for 3 weeks, increasing in intensity; recurrence of diarrhœa with pale offensive stools one week before admission. No pain. Moderate loss of weight.

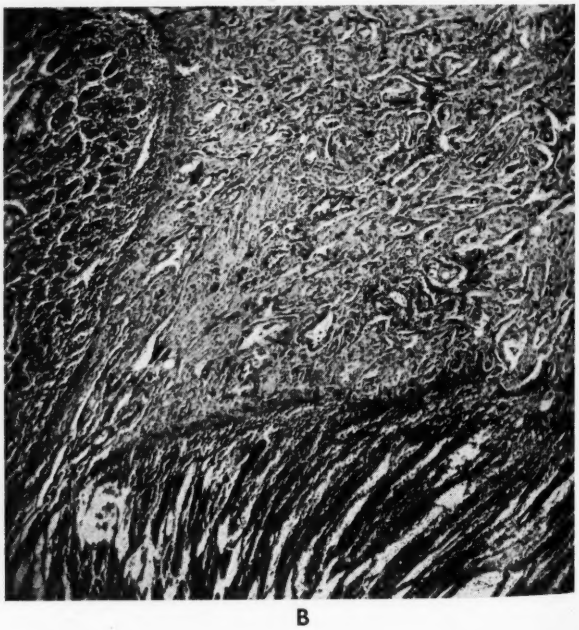
ON EXAMINATION.—A thin, emaciated and moderately jaundiced man. Liver edge 1½ in. below costal margin. Palpable enlargement of gall-bladder, and hard mass in region of head of pancreas. R.B.C. 4,800,000 per c.mm. Hb 90 per cent. Serum-bilirubin 4·8 mg. per cent. Prothrombin index 70 per cent. Hippuric acid test: normal.

Provisional diagnosis of carcinoma of head of pancreas.

OPERATION (July 24).—Under spinal anæsthesia. *One-stage radical pancreatico-duodenectomy with pancreatico-jejunostomy.* (Whipple, 1943 technique. *See Figs.* 155, I, 159, A.) The liver was a little enlarged, the common bile-duct and gall-bladder markedly so. There was a small hard mass localized to head of the pancreas. No secondaries detected. The portal and superior mesenteric veins were free. The duodenum and head of the pancreas were easily mobilized. The stomach was divided across the pyloric antrum and the common bile-duct above the duodenum. The pancreas was divided *across its neck*, the line of section being 1½ in. distant from the visible edge of the growth. The pancreatic duct was dilated, having a diameter of ¾ in. The jejunum was divided 4 in. from the duodenojejunal junction. The growth-containing mass was then separated from the portal vein and superior mesenteric vessels, and removed. The cut end of the jejunum was anastomosed to the cut end of the common bile-duct. The dilated pancreatic duct was then anastomosed to the



A

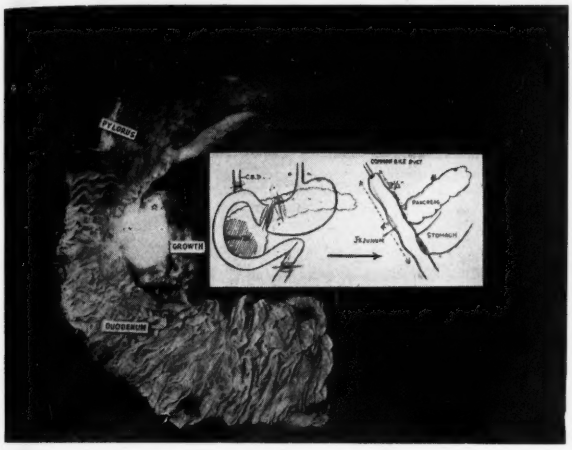


B

FIG. 158.—*Case* 2. Scirrhous adenocarcinoma of head of pancreas invading the duodenal wall. (× 35.)

side of the jejunum descending from its junction with the common bile-duct. Lower down the cut end of the stomach was joined to the side of the jejunum. The abdomen was closed without drainage.

PATHOLOGY.—"A section to include the whole head of the pancreas and adjacent duodenal wall shows a *well-differentiated Grade 1 adenocarcinoma of the pancreas* with marked scirrhous reaction (*Fig.* 159). The growth is just invading the submucosa of the duodenum but no farther. The growth, however, reaches the excision end, and may not have been completely removed."



A

B

FIG. 159.—*Case* 3. Scirrhous adenocarcinoma of head of pancreas.

PROGRESS.—In the immediate post-operative stage recovery was slow but steady. Jaundice cleared up, healing of wound *per primam*. Carbohydrate metabolism has been normal; the patient can tolerate and digest and absorb normal quantities of fat; but excessive intake immediately leads to the passage of 'oily' stools. He has gained weight and strength and is now back at his old job of house decorator and painter.

Comment.—Section through the neck of the pancreas seemed to be 1½ in. away from the near edge of the growth in the head, and yet the pathologist reports that the growth reaches the site of excision. This suggests the advisability of sectioning the pancreas farther to the left, through the body, as a routine measure in cases of carcinoma of the head.

Case 4.—**Adenocarcinoma of the head of the pancreas treated by a two-stage radical pancreatico-duodenectomy.**

Mr. H. B., aged 43, admitted on Sept. 21, 1945. Previous history of chronic gastric ulcer for which a partial gastrectomy with short loop post-colic anastomosis had been performed on Oct. 25, 1943. Present history of rigors for 6 weeks accompanied by increasing jaundice and pain in right hypochondrium. Some loss of weight. Pale stools and dark urine.

ON EXAMINATION.—Marked jaundice, little wasting. Tender and enlarged liver. Gall-bladder was not palpated. R.B.C., 4,900,000. Hb, 17 g. per cent. Serum-bilirubin, 6 mg. per cent. Van den Berg test, biphasic reaction. Hippuric acid test: poor excretion. Prothrombin index, 26·2 per cent. Prothrombin response to injection of vitamin K was prompt. Urine showed no excess of urobilinogen.

The picture was that of obstructive jaundice. Owing to the patient's poor general condition, operative treatment, if possible, should be staged.

FIRST OPERATION (Oct. 5, 1945).—Under nitrous oxide, oxygen, and ether. *Cholecystomy*. The liver was markedly enlarged; the gall-bladder and bile-ducts were dilated, and the former was tucked away under the liver. There was a hard nodule about 1½ in. in diameter in the head of the pancreas. There were no secondaries; the duodenum and head of the pancreas were easily mobilized and the veins appeared free. But the patient was in no fit state to stand a big operation. Cholecystostomy was done in favour of cholecyst-jejunostomy. Parenteral vitamin K was given. Recovery was rapid and the patient gained weight.

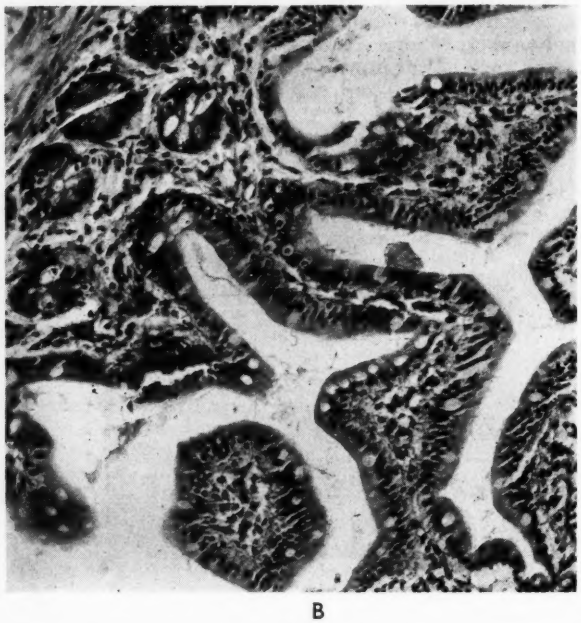
SECOND OPERATION (Oct. 22—3 weeks later).—Under spinal anæsthesia. *Radical pancreatico-duodenectomy*. The liver had shrunk to almost normal size. The pancreas was now generally enlarged and turgid, and the growth appeared to have invaded the whole head of the gland. All the neighbouring tissues were engorged with blood and the veins stood out. A radical excision of the head of the pancreas and duodenum was indicated, but obviously fraught with difficulties. After mobilization of the duodenum and head of the pancreas, the neck of the pancreas was cut across. The duodenum was then divided just to the right of the superior mesenteric vessel, and the cut end of the distal portion invaginated. On separating the head from the portal vein, the latter was torn and serious hæmorrhage occurred. The rent was eventually repaired by suture. Further separation led to more bleeding, so that eventually a narrow cuff of the head of pancreas had to be left in situ around the portal vein. The common bile-duct was divided and its end ligated and invaginated. By this time the patient's general condition was so poor that the operation had to be terminated quickly. The pancreatic stump was stitched, and the cholecystostomy was not disturbed. The continuity of the intestinal tract was, of course, assured by the previous partial gastrectomy. A drainage tube was put down to the pancreatic stump, and the abdomen closed.

PATHOLOGY.—"A whole section shows a Grade 2 adenocarcinoma of the pancreas invading the submucosa of the duodenum (*Fig.* 160). The papillomatous

growth in the ampulla and projecting into the duodenum through the orifice of the common bile-duct is a benign papilloma and does not appear to have any connexion with the carcinoma of the pancreas."

PROGRESS.—The immediate post-operative period was stormy. The patient developed a pancreatic fistula, which persisted for one week. Then signs of generalized acute peritonitis developed and the patient died from this 12 days after operation. A post-mortem examination was not made.

A



B

FIG. 160.—*Case* 4. Scirrhous adenocarcinoma of head of pancreas and simple papilloma of ampulla of Vater.

Comment.—It is the considered opinion of an experienced pathologist, who examined this specimen, that the papilloma of the ampulla was simple and that it bore no relationship to the adenocarcinoma of the head. Such an occurrence must be very rare indeed; and I know of no published report of a similar case.

Death in this case was indirectly caused by a pancreatic fistula; a complication which once again emphasizes the absolute necessity of anastomosing the pancreatic duct to the alimentary tract.

The extraordinarily rapid progress of the growth during the three weeks following the first operation caused some surprise and dismay at the second operation. Such a development is unfortunately not uncommon, and always leaves one in doubt as to how much of it is due to handling of the growth and disturbance at the first operation.

Case 5.—**Chronic pancreatitis simulating carcinoma of the head of the pancreas.**

Mr. A. B., aged 59 years, admitted July 10, 1944. History of painless progressive jaundice for 6 weeks, with clay-coloured stools and dark urine. Some loss of weight; but good appetite.

ON EXAMINATION.—Little wasting, marked jaundice. Liver and gall-bladder not palpable. R.B.C., 2,010,000 per c.mm. Hb, 44 per cent. W.B.C., 22,000. Blood-bilirubin, 3 mg. per cent. Hippuric acid test: normal. Prothrombin index, 73 per cent. Satisfactory and prompt prothrombin response to parenteral injection of vitamin K.

Five pints of blood were given to combat anæmia, and pre-operative vitamin K daily.

OPERATION (July 24).—Under ether anæsthesia. *Choledocho-jejunostomy*. Both head and body of the pancreas were hard and swollen. The liver was enlarged and bile-stained. The gall-bladder was congenitally absent, and common bile-duct dilated. Some enlarged lymph-nodes were present along the upper border of the body of the pancreas. The diagnosis was in doubt; and it was judged that a choledocho-jejunostomy and time would settle the diagnostic problem, and serve as a first stage to resection if the lesion should prove to be carcinomatous. The common bile-duct was divided above the duodenum; the lower end tied, and the upper end anastomosed to the jejunum, which was brought up in front of the transverse colon and omentum.

PROGRESS was satisfactory and rapid; jaundice cleared; and the patient began to gain weight and strength. A second operation was advised, but the patient refused to consider it.

He has now been at work for over a year, has gained more weight; and he is well, his appetite is good, and he can eat anything, including fatty foods. Examination of the abdomen on three occasions since operation has failed to reveal any abnormality.

Biopsy of a pancreatic lymph-node showed chronic inflammatory changes only.

Comment.—This was clearly a case of subacute or chronic pancreatitis completely simulating carcinoma, which cleared up after internal biliary drainage. It serves to illustrate the importance of performing the operation of radical pancreatico-duodenectomy in two stages in all cases in which the diagnosis of carcinoma is in doubt.

OTHER CASES

During the same period under review (1942-5, inclusive), the following additional cases were observed: 8 cases of carcinoma of the head of the pancreas (not confirmed histologically), 2 of the body, and 1 of the head and body. Of these only 2 cases of carcinoma of the head were operable; one died of liver failure 5 days after cholecyst-jejunostomy, and the other refused to submit to resection after preliminary cholecyst-jejunostomy and died 5 months later. A clinically palpable gall-bladder was found in 50 per cent of the cases with jaundice, which was present in 6 of the 8 cases of carcinoma of the head. In the remaining 2 cases the presenting feature was vomiting due to duodenal obstruction by growth. In all cases except 2, abdominal pain was a prominent feature, preceding the onset of jaundice.

SUMMARY

A short review of recent advances in the diagnosis and treatment of carcinoma of the head of the pancreas and ampulla-papillary region is presented.

The incidence of these tumours, and the difficulties and method of diagnosis are discussed. The evolution of surgical methods of treatment is traced from 1898 to the present, and an evaluation of present-day methods is made.

Detailed reports of 5 cases are given. One tumour, an adenocarcinoma arising from the papilla of Vater, was simply excised by the transduodenal route. Three carcinomas of the head of the pancreas were excised by radical pancreatectomy with one post-operative death. A case of chronic pancreatitis simulating carcinoma of the head of the pancreas is included to emphasize the diagnostic difficulties.

Acknowledgements.—Many of my colleagues have contributed in the investigation and treatment of these cases, and to them I am deeply grateful. I am especially indebted to my medical colleague, Dr. A. P. M. Page; to Dr. L. C. D. Hermitte, who has been untiring in his efforts with the reproduction of and reports on the specimens; and to Dr. Justin Davies, who gave the anæsthetics so skilfully. Finally, to the nurses and sisters in the wards and theatre it is a pleasurable duty to express my deep appreciation of their work and unstinted help.

REFERENCES

ALLEN, J. G. (1944), *Gastroenterology*, **3**, 490.
— — and JULIAN, O. C. (1942), *Arch. Surg.*, **45**, 691.
ASCHOFF, L. (1937), *Verh. dtsch. Ges. inn. Med.*, 261.
BAGENSTOSS, A. H. (1938), *Arch. Path. Lab. Med.*, **26**, 853.
BERK, J. (1941), *Arch. intern. Med.*, **68**, 525.
BRUNSCHWIG, A. (1937), *Surg. Gynec. Obstet.*, **65**, 681.
— — (1942), *Surgery of Pancreatic Tumours*. London: H. Kimpton.
CABOT, R. C., quoted by ELMAN, R. (1944), *Gastroenterology*, **3**, 417.
CATTELL, R. B. (1943, a), *Surg. Clin. N. Amer.*, **23**, 753.
— — (1943, b), In discussion paper of DRADSTEDT, L. R. (1943), *Ann. Surg.*, **118**, 576.
— — (1945), *New Engl. J. Med.*, **232**, 521.
CLUTE, A. (1936), *J. Amer. med. Ass.*, **107**, 91.
CODIVILLA, quoted by SAUVE, L. (1908), *Rev. Chir., Paris*, **1**, 113, 335.
COHEN, I., and COLP, R. (1927), *Surg. Gynec. Obstet.*, **45**, 332.
COLE, W. H., and REYNOLDS, J. T. (1945), *Surgery*, **18**, 133.
COLLER, F. A., and WINFIELD, J. M. (1934), *Amer. J. Surg.*, **25**, 64.
COURVOISIER, L. G. (1890), *Statistisch Beitrage zur Pathologie und Chirurgie der Gellenwege*, 58. Leipzig: Vogel.
DAM (1935), *Biochem. J.*, **29**, 1273.
DICK, J. C. (1939), *Brit. J. Surg.*, **26**, 757.
DRAGSTEDT, L. R., DRAGSTEDT, C. A., MCCLINTOCK, J. T., and CHASE, C. S. (1918), *Amer. J. Physiol.*, **46**, 584.
DRAPIEWSKI (1944), *Amer. J. clin. Path.*, **14**, 549.
EUSTERMAN, G. (1922), *Trans. Amer. gastro-ent. Ass.*, **25**, 126.
EWING, J. (1928), *Neoplastic Disease*, 3rd ed. London: Saunders.
FRASER, Sir JOHN (1938), *Brit. J. Surg.*, **26**, 393.
GRAY, H. K., and SHARPE, W. S. (1943), *Surgery*, **14**, 831.
GORDON-TAYLOR, Sir GORDON (1942), *Brit. med. J.*, **2**, 119.
HALSTED, W. S. (1899), *Boston med. surg. J.*, **141**, 645.
HANGER, F. M., and GUTMAN, A. B. (1940), *Jour. Amer. med. Ass.*, **115**, 263.
HIRSCHEL, G. (1914), *Münch. med. Wschr.*, **61**, 1728.
HUNT, V. C. (1940), quoted by BRUNSCHWIG, A. (1942).
— — (1941), *Ann. Surg.*, **114**, 570.
— — and BUDD, J. W. (1935), *Surg. Gynec. Obstet.*, **61**, 651.
IVY, A. C., and ROTH, J. A. (1943), *Gastroenterology*, **1**, 655.
JUDD, E. S., and PARKER, B. R. (1928), *Arch. Surg.*, **17**, 1.
KAUSCH, W. (1909), *Z. Chir.*, **36**, 1350.
KORTE, W. (1909), *Arch. klin. Chir.*, **89**, 1.
LIEBER, M., STEWART, H., LUND, H. (1939), *Ann. Surg.*, **109**, 219.
LORD, J. W., and ANDRUS, W. D. (1941), *Arch. intern. Med.*, **68**, 199.
MANN, F. C. (1942), Discussion of paper by MATEER, J. G., et al., *Amer. J. Dig. Dis.*, **9**, 13.
— — and KAWAMURA, K. (1919), *J. Amer. med. Ass.*, **73**, 878.
MAYO-ROBSON, A. (1900), *Int. Cong. Med., Paris*, **10**, 140.
MICHAUX (1906), quoted by BRUNSCHWIG, A. (1942),
MOORHEAD, J. J., and LANDES, H. E. (1919), *J. Amer. med. Ass.*, **72**, 1127.
MOYNIHAN, B. (1905), *Abdominal Operations*. London.
OLWIN, J. G. (1941), *Arch. Surg.*, **43**, 633.
ORR, T. G. (1945), *Surgery*, **18**, 144.
OUTERBRIDGE, G. W. (1913), *Ann. Surg.*, **57**, 402.
PALLIN, G. (1920), *Beitr. klin. Chir.*, **121**, 84.
PERRY and SHAW (1892–3), *Trans. Path. Soc.*, 84.
POPPER, H. (1936), *Wien. klin. Wschr.*, **49**, 207.
QUICK, A. J., STANLEY-BROWN, M., and BANCROFT, F. W. (1935), *Amer. J. med. Sci.*, **190**, 501.
RANSOM, H. K. (1938), *Amer. J. Surg.*, **40**, 264.
RIVES, J., ROMANO, S., and SANDIFER, F. (1937), *Surg. Gynec. Obstet.*, **65**, 164.
RUDDOCK, J. C. (1937), *Ibid.*, **65**, 623.
SALLICK, M. A., and GARLOCK, J. H. (1942), *Ann. Surg.*, **115**, 25.
SCHNEDORF, J. G., and ORR, T. G. (1941), *Ann. Surg.*, **114**, 603.
SHERLOCK, SHEILA (1945), *Lancet*, **249**, 397.
STEIGMANN, F., and POPPER, H. (1943), *Gastroenterology*, **1**, 645.
— — and DYNIEWICZ, J. M. (1943), *Ibid.*, **1**, 743.
TENANI (1922), *Policlinico*, **39**, 291.
TUFFIER (1905), quoted by BRUNSCHWIG, A. (1942).
WALTERS, W., and DEHNE, E. (1932), *Surg. Gynec. Obstet.*, **54**, 832.
WHIPPLE, A. O., PARSONS, W. B., and MULLINS, C. R. (1935), *Ann. Surg.*, **102**, 763.
— — (1940), quoted by BRUNSCHWIG, A. (1942).
— — (1941), *Ann. Surg.*, **114**, 612.
— — (1942), in Discussion, HARVEY, S. C., and OUGHTERSON, S. W. (1942), *Ann. Surg.*, **115**, 1085.
— — (1943), in Discussion of paper of DRAGSTEDT, L. R. (1943), *Ann. Surg.*, **118**, 576.
— — (1945), *Ann. Surg.*, **121**, 847.

THE EXCISION AND REPAIR OF DEEP THERMAL NECROSIS

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THE surgical excision of tissues irreparably damaged by heat has been advocated for a considerable time (Wilms, 1901; Willis, 1925; Mason, 1941; Young, 1942), but the method has several limitations which may have prevented its practice in suitable cases.

In theory, the sooner a devitalized area is separated from its host, the sooner will surgical repair be practicable and the shorter the stage of toxic absorption from the infected slough. This is generally accepted, but we have found less support for our opinion that the corollary of this statement is that, whenever possible, excision of the entire area should be carried out before proteolysis occurs and should be immediately followed by primary repair. In most cases such repair can be adequately effected by split skin-grafts providing these will have a vascular bed for their reception; exposed tendons or cortical bone, for example, cannot support any thickness of free graft whatsoever; their failure over such areas will result in further infective necrosis.

For primary excision to be practicable, the following must be fulfilled: (1) The area of necrosis must be capable of total excision. (2) The area to be excised must not be surrounded by an area of partial-thickness skin loss. (3) Facilities for immediate repair of the area must be available.

To consider these items in detail:—

1. *The Area of Necrosis must be capable of Total Excision.*—Although partial excision of the slough may diminish the degree of infective toxæmia, it is of limited value in that epithelialization will be delayed until separation of the remaining slough has occurred. The more common difficulty, however, is the determination of whether or not deep epithelial elements have survived. We consider that if the other criteria can be fulfilled, the sacrifice of a few sweat glands and hair follicles is of no importance. In a large burn where, possibly, not more than 10 per cent of the deep elements are viable, the rate of healing from these survivors is so slow as to be valueless; scar contracture will occur in the intervening areas, producing, ultimately, delay in epithelialization and probable deformity. It is the difficulty of estimating the proportion of surviving elements which limits the field for early surgery; this is in no way simplified by the knowledge that a proportion of those surviving the original heat necrosis will fail to survive the infective phase. The use of picro-fuchsin to determine the proportion of viable structures in a burn has been investigated by Patey and Scarff (in 1944). We have had no experience of this method, nor of the use of fluorescein, which has been used by American workers. Neither of these methods gives any estimate of which cells will fail to survive the infective phase, and it is clear that this is entirely a matter of chance.

2. *The Area to be excised must not be surrounded by an Area of Partial-thickness Skin Loss.*—Although we have excised areas of full-thickness skin loss, when surrounded by a more superficial loss, we do not consider this a valuable technique unless performed in the second or third week, when the area of slough is clearly demonstrable. The grafting of such defects is hazardous, as the inevitable infection of the surrounding areas will tend to diminish the percentage take of the graft.

We regard this as one of the most important criteria to be fulfilled before early operation upon a thermal burn is contemplated.

3. *Facilities for Immediate Repair of the Area must be available.*—Immediate repair of the defect will, in most cases, mean the use of partial-thickness skin-grafts immediately following the excision. Any delay, even for a few days, between the two parts of the operation may permit local sepsis to occur, and will allow the development of scar tissue in the recipient areas. Even with the aid of chemotherapy it is preferable to graft a recent surgical wound rather than granulations, however admirable (Rank, 1940; Mowlem, 1941, 1944).

Other factors bear upon the question of immediate repair; the availability of adequate donor areas for the split skin-grafts and the technical equipment of the surgeon to cut these grafts without risk of damaging important donor areas, are but two essential requisites.

While these three criteria are, clearly, not absolute, when they occur together we consider that the early excision and immediate repair of an area of complete thermal destruction should be considered.

The case quoted to illustrate this active approach cannot be considered as typical of the type of loss suitable for surgery; nevertheless, we find it difficult to believe that any other method of treatment would, in the presence of such extreme destruction, have enabled the patient to stand upon her own limbs two months after injury.

CASE REPORT

Jan. 4, 1945: Mrs. B., aged 47, was blown by the explosion of a V2 bomb into her own fire and was pinned by masonry for 3 hours in more or less direct contact with the embers.

Jan. 6: Transferred to an E.M.S. Plastic Unit. Condition on admission good; comfortable and uncomplaining. (*Fig.* 161.)

The right leg had been burnt around the circumference of the distal half of the thigh and proximal half of the leg. Diagnosis of the depth of the burn was complicated by the liberal application of gentian violet dye prior to admission.

The left leg showed an extreme degree of heat coagulation; the muscles of the thigh and calf on the lateral aspect were grey and quite friable and had the appearance of slowly-cooked meat.

Jan. 12: OPERATION. Necrotic skin was removed from the lateral and posterior aspects of the thigh from the gluteal fold to the popliteal fossa. In the leg, the skin of the upper two-thirds of the posterior and anterolateral aspects was excised, together with that over

the upper two-thirds of the subcutaneous border of the tibia.

In the thigh the lateral half of the biceps was removed, while in the leg the upper two-thirds of the muscles in the antero-lateral compartment were excised completely,

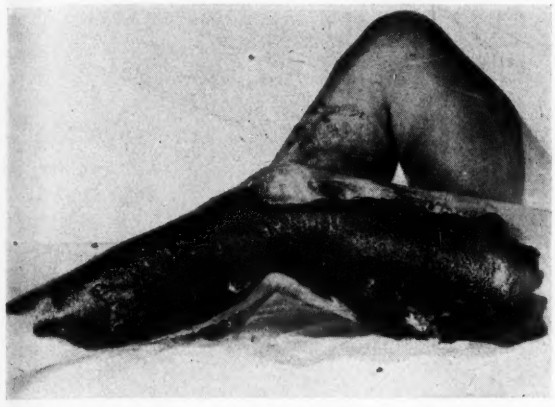

FIG. 161.—(Jan. 6, 1945.) Condition on admission, showing the black necrotic skin with muscle protruding in the calf.

as were the peroneal nerve and the lateral half of the calf muscles. All these structures were quite necrotic.

Operation was then terminated, as the patient's general condition was deteriorating and the circulation of the foot had suffered when the anterior tibial artery had been tied, as it perforated the interosseus ligament.

It was known that all necrotic tissue had not been removed at this operation and that many areas, though not necrotic, were markedly affected. Further ablation was clearly necessary as the upper two-thirds of the fibula, its interosseus membrane, and the anterior half of the tibia were exposed and would sequestrate.

Penicillin-sulphonamide powder was insufflated on the wound, a pressure dressing applied, and the limb immobilized in plaster.

FIG. 162.—(Jan. 24.) After excision of avascular skin and muscle. Tibia and fibula exposed.

An interesting note on the pathology of heat necrosis was the marked absence of bleeding from veins, although arterial pulsation was undiminished. In some areas (the upper half of the anterior tibial, for example) the local arteries ran apparently unscathed through muscle so friable that it could be removed with the finger.

Jan. 14: Post-operative condition satisfactory.

Jan. 16: Suddenly, after complaining of severe pain in the foot, the patient became quite irrational, with pyrexia, marked tachycardia, and a raised respiratory rate. 1½ pints of blood were given, with improvement.

Jan. 17: OPERATION. Further small areas of slough were removed, mainly fascia. Some muscle bundles in unopened and apparently viable fascial sheaths were, in fact, found to be necrotic. These were excised. In the peroneal region clean granulations were present over a small area. Dressing as before.

Jan. 22: Considerable improvement following operation. Toxic facies no longer present.

Jan. 24: OPERATION. At least 95 per cent of the area was found to be covered with clean granulations. The remaining 5 per cent consisted of odd tags of sloughing fascia, skin, and muscle. These were removed. The cortex of the antero-medial and antero-lateral aspects of the tibia in its upper two-thirds, together with the crest and most of the tibial tubercle, was

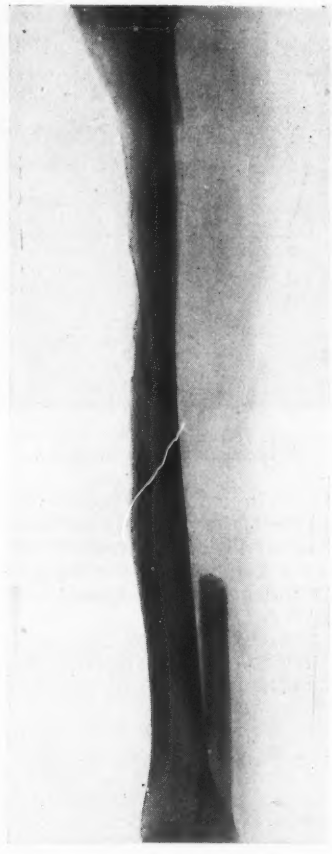

FIG. 163.—(Feb. 10.) Radiograph showing the amount of tibia and fibula removed.

removed by hammer and chisel to expose the medulla. The upper two-thirds of the fibula and interosseus membrane were removed. Strips of thin split-grafts cut from the abdomen were used to cover most of the leg and a small area of thigh. Pressure dressing and plaster fixation. Neither penicillin nor sulphonamide powder was used. (*Fig.* 162.)

Feb. 2: OPERATION. Further cortex was removed from the tibia, the amount removed at the previous operation being inadequate in that none of the grafts over this area had survived and granulations were scanty and of poor quality. 50 per cent of the grafts used at the previous operation appeared to have survived. Granulations between these grafts were clean and of good texture. Further grafts were cut from both thighs to give about 30 per cent cover over the calf and 75 per cent over the thigh.

Feb. 16: OPERATION. Further grafts, cut from the back, were used to cover the remaining defects. Grafts from previous operation 100 per cent. (*Fig.* 164.)

Feb. 27: Left leg almost completely healed, areas remaining unhealed being of no significance. Afebrile for one week. Started to get up; legs very weak, but without deformity of knee or ankle. General condition excellent. This was 7 weeks after injury.

The patient has since been seen on various occasions. She walks well with a stick and a toe spring to make good the loss of the muscles of the anterior tibial and peroneal groups. Foot-drop has not occurred owing to the fixation of the distal muscle stumps by scar tissue; because of this there is only a few degrees of plantar flexion; passive dorsiflexion full. Œdema of the foot is controlled by a crêpe bandage; sensation on the sole normal. Knee flexion 75°. No further surgery required.

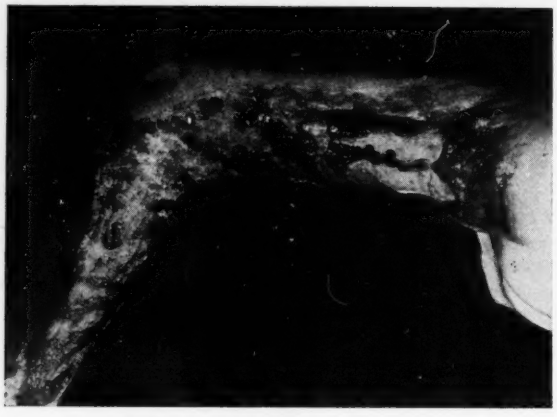

FIG. 164.—(Feb. 16.) After grafts had been used on two occasions to cover the defects.

Comment.—Three further points of interest arise from this case; first, that the only occasion in which the patient was severely ill, or suffered toxæmia in any way comparable to the magnitude of the burn, occurred when the surgical treatment was inadequate, and was relieved by further surgery.

Secondly, that chemotherapy was used on two occasions only and that on the first of these it could not compensate for inefficient surgical treatment. Penicillin in this type of case should be used locally in conjunction with surgery; systemically, should a cellulitis or lymphangitis develop as a result of surgery.

Thirdly, attention is drawn to the variable sites of donor areas for the split-skin grafts. At no time was the repair delayed by lack of available donor areas.

In this case it will be noted that immediate repair of the area was not performed. This was because the extreme degree of loss made total excision of the affected area at one operation impossible.

SUMMARY

1. The early excision and repair of thermal burns involving destruction of all layers of the skin is discussed.

2. The circumstances in which this is the method of choice are itemized.

3. An illustrative case is described.

4. The word 'burn', when used in this paper, is used for lack of a suitable alternative. We consider the mechanism of tissue death, whether it be traumatic, chemical, thermal, or electrical, to be of academic interest only; our surgical interest must be toward the repair of the loss.

FIG. 165.—(April 11.) Final condition of leg. Walking well with toe spring. Scars surrounding the graft are hypertrophic, but already subsiding. No further treatment required.

My grateful thanks are due to Mr. Rainsford Mowlem, Surgeon-in-Charge, Plastic and Jaw Unit, Hill End Hospital, St. Albans, for advice and assistance.

REFERENCES

BROWN, J. B., and MCDOWELL (1943), *Skin Grafting of Burns*. J. B. Lippincott Co.

MASON, M. L. (1941), *Surg. Gynec. Obstet.*, **72**, 250.

MOWLEM, R. (1941), *Proc. R. Soc. Med.*, **34**, 337.

— — (1944), *Ibid.*, **37**, 215.

PATEY, D. H., and SCARFF, R. W. (1944), *Brit. J. Surg.*, **32**, 32.

RANK, B. K. (1940), *Brit. med. J.*, **1**, 846.

WILLIS, A. M. (1925), *J. Amer. med. Ass.*, **84**, 655.

YOUNG, FORREST (1942), *Ann. Surg.*, **116**, 445.

RECONSTRUCTIVE SURGERY OF THE HAND
WITH SPECIAL REFERENCE TO DIGITAL TRANSPLANTATION

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THE experience gained while in charge of the Hand Clinic at Leith Hospital during the past four years has convinced me that, if the ideal of treatment is to be approached, there must be continuity in a single unit. In dealing with a mutilating injury of the hand, the full treatment which may be required can be divided into five aspects: primary reparative surgery; physiotherapy; reconstructive surgery; restoration of maximal function; and vocational training. These aspects are not clear-cut, nor does the need for each exist in every case. The present paper deals only with the stage of reconstructive surgery.

METHODS OF RECONSTRUCTION

Crippled hands following severe injury fall into two main groups: those in which structures are present but lack function; and those in which structure has been reduced to a functionless level, as when all digits have been lost.

In the first of these, it is usually apparent to both the patient and the surgeon that something should be done to improve matters; in the second, it is not so obvious, and there may be a danger of such unfortunate patients being allowed to leave hospital without having been fully treated, to struggle along as best they can. It is not the purpose of this paper to go into the intricacies of the procedures which may be employed to restore function to the functionless digit, but to demonstrate that much can be done for the second type of case—that is, where insufficient functional units remain.

The essential anatomical structures which subserve the basic function of the hand are two digits capable of being brought into opposition with each other; in other words, a thumb and one finger. There is little to choose between a hand which has a thumb but no opposing finger and one which consists of four fingers but no thumb; the function of both has been very considerably reduced. The aim in reconstruction should be to restore to these mutilated hands at least the basic finger-thumb combination, and to achieve this aim it is obvious that one missing digit must be replaced. To restore a digit to the site of one which has been lost, three methods are available: first, the transposition of one of the remaining digits of the same hand; secondly, a whole finger graft from the other hand (a toe has been used quite successfully, but there can be little doubt that a finger gives the better functional result); and, thirdly, the manufacture of an artificial finger from living tissues. The transposition of a finger from the same hand has its main use in restoring the loss of a thumb: a further rather unique use is described below. A whole-finger graft from the uninjured hand finds its main field in the case of total loss of all four fingers. The third procedure —plastic construction of a finger of the fixed prop type—has several disadvantages: mobility is absent, sensation is absent or only partly present, viability of the finger is somewhat precarious and the partially insensitive finger is liable to be injured or burned. The time spent in its construction is longer than that for the other methods, more operative stages are required, and at least two additional scars are made. With these disadvantages, it is preferable, if possible, to use either of the first two methods. In cases where this is not possible, the third method is indicated, and patients who have undergone this type of reconstruction have been very grateful.

Six cases are reported: two examples of each of the three methods of reconstruction.

CASE REPORTS

TRANSPOSITION OF A FINGER OF THE SAME HAND

Case 1.—J. R., aged 21. This man had his thumb avulsed in a rope-making machine. The base of its metacarpal remained and the carpo-metacarpal joint was uninjured (*Fig.* 166).

FIG. 166.—*Case* 1. Showing total loss of left thumb.

OPERATIVE TECHNIQUE.—Incisions were made on the palmar and dorsal aspects of the hand, similar to those for a syndactyle. First, the digital nerve and artery to the adjacent sides of the index and middle fingers were exposed from the palmar aspect. In order that the index finger could be widely separated from the rest of the hand without damaging or destroying its nerve or arterial supply, the palmar digital nerve was split from its bifurcation to its origin from the median, and the branch of the palmar digital artery to the radial side

of the middle finger was divided between ligatures (*Fig.* 167). On the dorsal aspect, an oblique band joining extensor indicis to the extensor of the middle finger was divided at the lower margin of the extensor retinaculum.

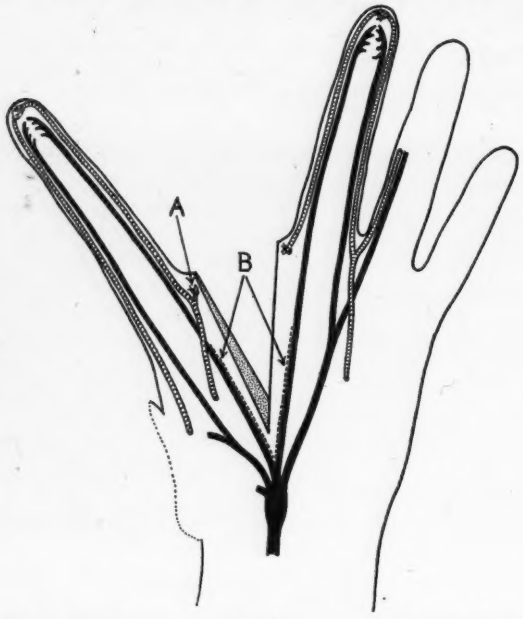


FIG. 167.—Same case. Illustrating method of maintaining bilateral nerve and arterial supply. A, Division of digital artery to radial side of middle finger. B, Split portion of palmar digital nerve, indicated by interrupted line.

The second metacarpal with its periosteum was then sectioned ½ in. from its base, care being taken not to injure the radialis indicis artery. The dissection was resumed on the palmar surface, and access to the cellular plane between the palmar and dorsal interosseous muscles was gained after dividing the transverse carpal ligament. The palmar interosseous muscle was separated and retracted to expose that portion of the second dorsal interosseous muscle arising from the ulnar side of the second metacarpal. This origin was stripped from the bone. The index finger was now completely separated from the rest of the hand as far as the base of its metacarpal. It was structurally intact in so far as it had a bilateral nerve and arterial supply, flexor and extensor tendons, its lumbrical and interossei. The whole unit was then moved radially, rotated through 90°, and fixed by suturing the second metacarpal to the stump of the first metacarpal. Next, the distal end of the extensor tendon was sutured to the stump of extensor pollicis longus.

The wound was closed by suturing the dorsal flap round the third metacarpal and bringing the palmar flap round the new thumb. This flap was not long enough to close completely the gap and a small skin-graft was applied. An amputation through the proximal interphalangeal joint of the transposed digit was then performed. *Fig.* 168 show the final result.

Case 2.—A. C., aged 14½. This boy caught his right hand between the rollers of a baking machine, and sustained a 'degloving' injury. The skin had been divided 1 in. above the proximal flexion crease of the wrist, and turned inside out to the level of the metacarpo-phalangeal joints.

OPERATIVE TECHNIQUE.—

1. *Initial Operation.*—This operation was performed within three hours of the injury. After the usual surgical toilet, all fat was excised from the skin-flap, which was then replaced and sutured in position as a full-thickness pedicle graft. Several small incisions were made for the purpose of drainage, and a pressure dressing was applied; but later the whole of the graft sloughed, and the index, ring, and little fingers became gangrenous (*Fig.* 169). This gangrene was ischæmic in nature and was probably due to arterial thrombosis following trauma to the main vessels. Though not all of the middle finger died, necrosis and separation of the whole of the proximal phalanx occurred, leaving the finger attached by a small soft-tissue pedicle containing one digital artery.

2. *Skin-grafting.*—A tubed pedicle graft was raised from the skin of the right iliac fossa, in an oblique direction from the anterior superior iliac spine towards the left shoulder. All dead tissue was excised from the hand, which was treated in closed plasters till a healthy granulating surface was obtained.

Five weeks later the distal end of the pedicle was detached from the abdomen, opened out, and applied

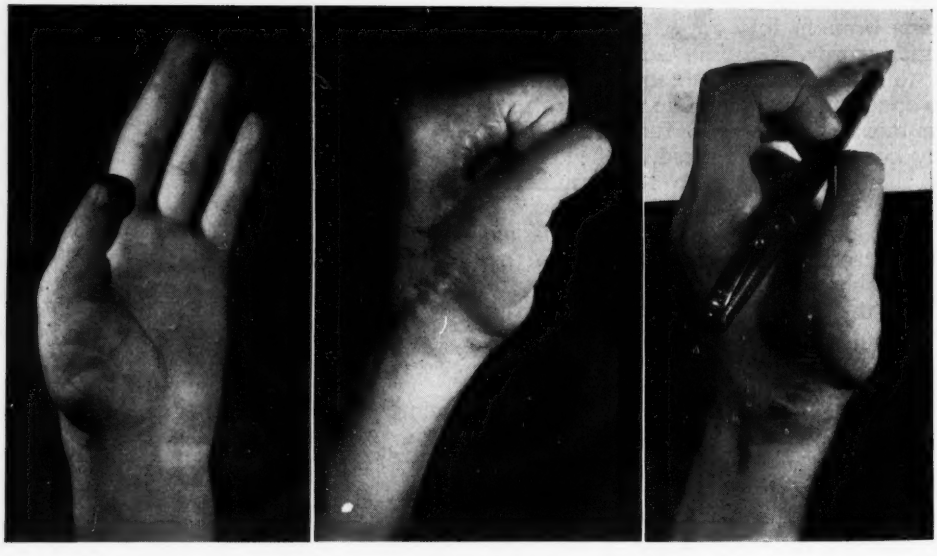


FIG. 168.—Same case. Showing hand after transposition of index finger to site of missing thumb, with amputation of distal two phalanges.

to the dorsal surface of the wrist, the free end being sutured to the proximal skin edge. At the same time, the tube was extended towards the costal margin. Two weeks later the extended portion of the pedicle was raised and applied to the dorsum of the hand, and a further extension of the tube over the costal margin was carried out. At the end of a further two weeks, the pedicle was detached from the chest and carried over the metacarpal heads to cover the distal portion of the palm. Three weeks later, a split-skin dermatome graft was used to cover the remaining raw area of the palm and wrist.

FIG. 169.—*Case* 2. Seven days after injury; the necrotic skin has been cut away. Dry gangrene of index, ring, and little fingers, most noticeable at nails.

FIG. 170.—Same case. After completion of skin-grafting. The dorsum and distal portion of palm are covered by an abdominal pedicle graft, the wrist and proximal portion of palm with a split-skin graft. The middle finger is flail and lying towards the ulnar side.

By the time healing was complete, the thumb had become somewhat adducted and a flexion deformity of the wrist was present. The middle finger maintained its viability, but it was flail and without active movement (*Fig.* 170).

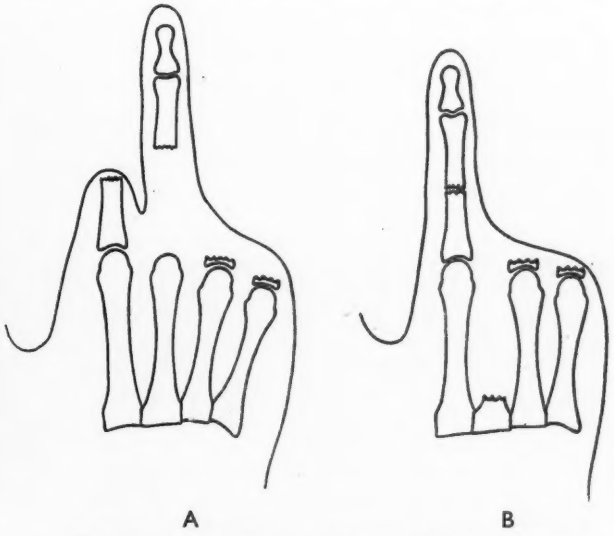
After a period of physiotherapy, during which time movements of the thumb and wrist improved, the reconstruction of a 'forceps hand' was undertaken. The middle finger had lost only its proximal phalanx. The index finger had lost all but its proximal phalanx. The middle finger was incapable of movement owing to the loss of its interossei and lumbrical attachments and the destruction of its long tendons. The index stump was capable of movement by virtue of its lumbrical, interossei, and long tendons which were adherent to the proximal phalanx (*Fig.* 171, A). It was reasoned that, if the remnants of these two fingers could be combined, one whole and functioning digit would result.

3. *The Reconstruction.*—

Middle finger: A triangular incision, its apex distally, was made on the radial side of the proximal phalanx and the enclosed area of skin excised. An accommodation space was made in the fibrous-tissue core of the finger, extending distally to the base of the middle phalanx. The whole of the middle metacarpal was then excised through a separate incision on the dorsum of the hand.

Index stump: The whole of the skin and subcutaneous tissue was excised with the exception of an inverted V-shaped area on the radial side. Fibrous tissue was cleared from the end of the phalanx till healthy bone was exposed.

The middle finger was moved towards the radial side till the proximal phalanx of the index finger occupied its accommodation space. This lateral shift was made possible partly by the mobility of the soft tissues and partly by the movement of the fourth and fifth metacarpals towards the bed of the excised third metacarpal (*Fig.* 171, B). The proximal phalanx of the index finger was then sutured with catgut to the second phalanx of the middle finger. The margins of the two skin incisions which had now come together were sutured and plaster-of-Paris fixation employed.



A B

FIG. 171.—Same case. A, Showing index stump and absence of proximal phalanx of middle finger. B, Showing transposition of middle finger to index site. The middle metacarpal has been excised. The fourth and fifth metacarpals have been moved towards the radial side.

4. *Further Operative Procedures.*—Three months after operation no bony union had occurred. Following a further operation at which the bone ends were freshened and sutured with stainless steel wire, firm union occurred in six weeks. As there was still considerable adduction

of the thumb and flexion of the wrist, a Z-plastic of the first interdigital cleft and lengthening of the flexor tendons in the wrist were carried out. At the same time, the rather voluminous abdominal graft was undercut, trimmed, and re-sutured. *Fig.* 172 shows the result six months after the completion of this operation.

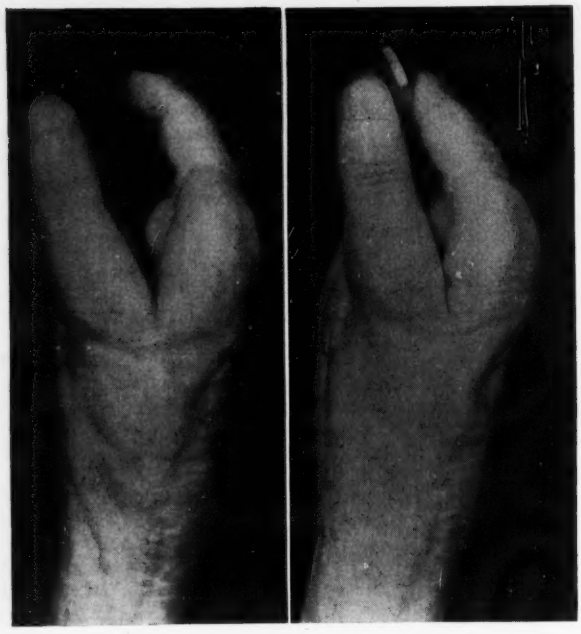


FIG. 172.—Same case. Showing result of transposing middle finger to site of index.

Comment.—Both these patients are now fully employed. J. R. (*Case* 1) has returned to his rope works and, though not operating the same high-speed machine, is earning the same wage as before his accident. A. C. (*Case* 2) has found employment in the boot-making trade. Using his ' forceps hand ', he can pick up and place in position the small sprigs used in soling shoes. He drives them with a hammer held in his left hand.

In *Case* 1 the distal portion of the finger was amputated at the time of operation as it was thought that the finger would be too long and cumbersome. (The thumb is normally two phalanges shorter than the index finger.) It was later considered that this was a mistake. The value of a nail and so specialized an area as the finger pulp is too great for them to be sacrificed for appearance and stability. Although the base of the first metacarpal, carrying the insertion of the long abductor tendon, was present, it was not necessary for the success of the operation. A fibrous joint between the transplanted metacarpal and the recipient carpus is quite satisfactory, provided the tendon of abductor pollicis longus is drawn down and fixed to the metacarpal. The direction of pull of the flexor tendons creates an adduction force which, if unopposed by an abductor, will draw the finger away from its all-important position of opposition to the middle finger (now the index finger).

It will be recalled that in *Case* 2 non-union with displacement of the bone ends occurred after the finger had been transplanted, and yet rapid union followed a subsequent operation at which the bone ends were freshened and resutured. Two possible reasons for this are suggested. First, the blood-supply of the finger following its transplantation must have been very poor, and vascular insufficiency may have been a direct cause of the failure to unite. Secondly, the bone suture at the time of the transplant was performed with catgut, and at the subsequent operation with stainless steel wire. Wire ensures a more adequate and lasting fixation than catgut with less tissue reaction; in no case where I have used stainless steel wire for bone suturing has delayed union or displacement of the fragments occurred.

Case 2 illustrates the extreme importance of maintaining the position of function of the hand throughout all stages of treatment. During the skin-grafting stage the thumb became adducted and the wrist flexed. These deformities were produced by the position of the hand as it lay on the body surface for a period of four weeks. The possibility of such deformities occurring was foreseen from the outset and attempts were made to prevent them by various means, including moulded perspex splints, but the attempts were, for the most part, unsuccessful.

WHOLE FINGER GRAFT FROM THE OTHER HAND

Case 3.—W. B., aged 15. This lad had his right hand caught in a mincing machine and lost the greater part of all four fingers (*Fig.* 173).

FIG. 173.—*Case* 3. Amputation of all four fingers in a mincing machine.

OPERATIVE TECHNIQUE.—

1. *Initial Operation.*—It was decided at this primary operation that a whole finger graft from the other hand should be performed at a later date, and the index stump was chosen as the recipient site. As conservation of the length of this stump meant much to the success of the future transplant, a minimum of bone excision was carried out though this meant suturing the flaps under considerable tension.

2. *The Finger Graft.*—The general plan of this stage was as follows: to divide all structures of the ring finger of the left hand with the exception of a soft-tissue flap on the ulnar side, this flap containing the digital artery and nerve; to swing the distal segment of the finger ulnarwards through an angle of 90° and oppose

its raw end to the prepared end of the right index stump (*Fig.* 174); to suture the bone, the tendons, and the digital nerve on the exposed side; and finally, to cover

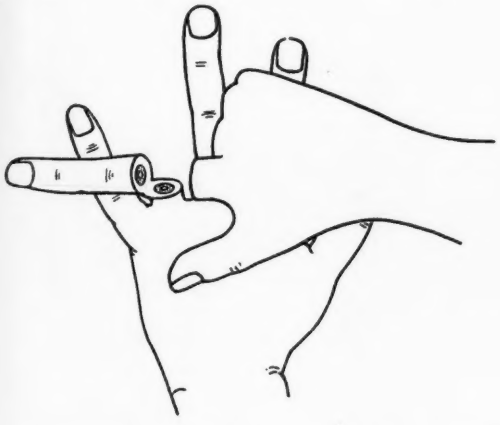


FIG. 174.—Same case. Showing left ring finger swung on a neurovascular pedicle through 90° and ready to be opposed to end of right index stump.

all raw areas and allow the fingers to grow together. If all structures were to be divided and sutured at the same level, there would be a danger of the formation

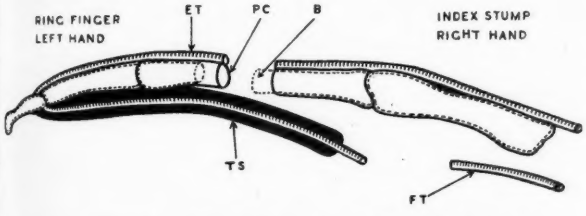


FIG. 175.—Same case. Illustrating level of division of similar structures of right index and left ring fingers. ET, Extensor tendon; B, Naked bone end; TS, Tendon-sheath; FT, Flexor tendon; PC, Periosteal cuff.

of a through-and-through block of scar tissue incorporating all suture lines and seriously interfering with both blood-supply and function. To minimize this danger, each structure was divided at a different level in the following manner:—

Right index finger (*Fig.* 175): The stump was first cleanly guillotined as near to its distal end as possible; then, working proximally through a lateral incision on its radial side, the periosteum and the extensor expansion were incised round the full circumference 1 cm. from the guillotined surface. The portion of the periosteum distal to this incision was elevated and removed, leaving

FIG. 176.—Same case. Photograph taken at operation after suture of bone, periosteum, and tendons.

a length of 1 cm. of naked bone protruding beyond the periosteal edge. The digital nerves were divided midway between the end of the bone and the end of the periosteum. The adherent and fibrous flexor tendon was dissected free and divided through healthy tendon tissue in the palm above the tendon-sheath.

Left ring finger (*Fig.* 175): Here the incision passed round the base of the finger but did not completely encircle it on the ulnar side. Through a lateral incision on the radial side, proximal to the first incision, the periosteum, together with the extensor tendon, was divided 1 cm. proximal to the level of the circular skin incision; it was then elevated in the form of a cuff distally for a distance of 1 cm. At this level the bone was sectioned and the periosteal cuff allowed to fall back. The digital nerve on the radial side was, as before, divided midway between the bone and periosteal ends. By careful dissection the fibrous sheath containing the flexor tendons was stripped from the base of the proximal phalanx and the neck of the metacarpal, and the tendons

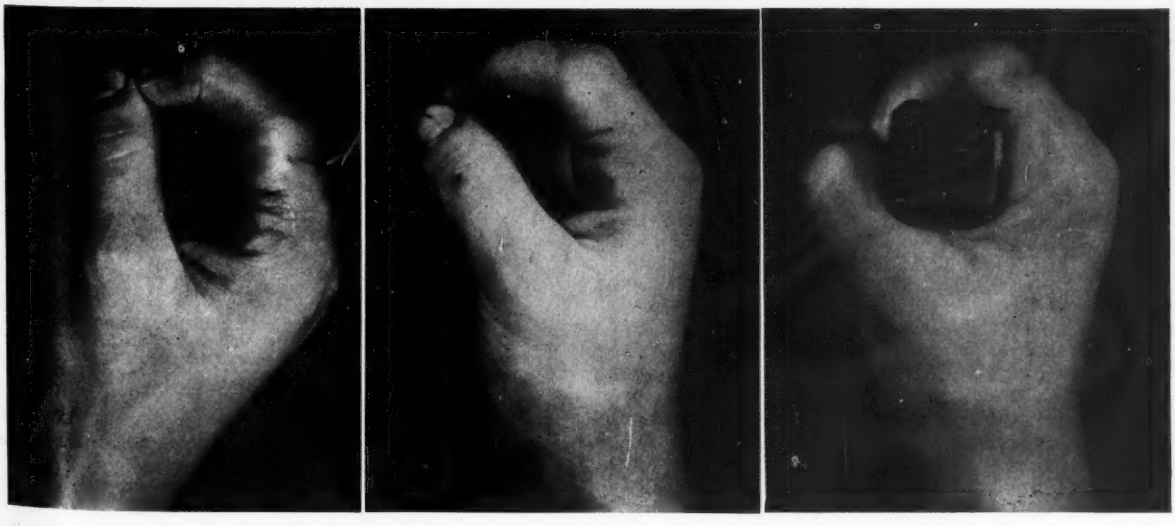
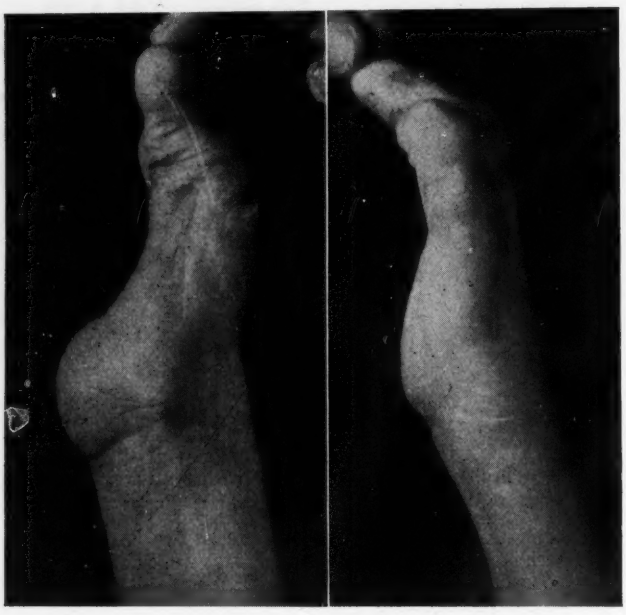
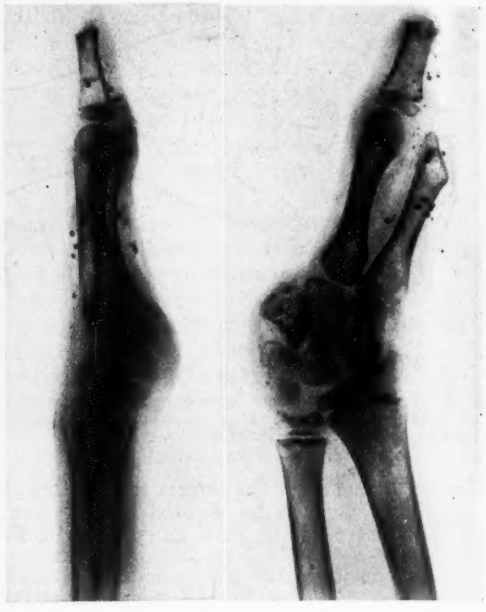


FIG. 177.—Same case. Showing end-result of a whole finger graft from uninjured hand.

divided in the palm above the proximal end of the sheath. Access to this latter part of the dissection was gained through a separate incision in the palm.

The hands were crossed and the forearms bound into a plaster shell which had been prepared some days





FIGS. 178, 179.—*Case* 4. Anti-personnel bomb injury to right hand. Numerous bomb fragments are seen in the radiograph.

previously. The distal portion of the left ring finger was swung on its neurovascular pedicle through an angle of 90° and its end brought into apposition with the end of the right index stump. The periosteal cuff of the ring finger was slid over the naked bone end of the index stump and all divided structures came together end to to end. Stainless steel wire was used throughout in suturing the flexor digitorum profundus tendon, bone, extensor expansion, and digital nerve, in that order (*Fig.* 176). The wound was closed by first suturing the opposed edges of the two circular incisions. This left the proximal edge of the circular incision in the ring finger and the longitudinal incisions on the radial side of each finger to be dealt with. The longitudinal incision in the ring finger was closed completely while that in the index stump was closed in its proximal two-thirds, the distal one-third being left open. By retracting the edges of this terminal portion of the incision, a roughly semi-circular edge was obtained. This edge was sutured to the free edge of the circular incision in the ring finger.

Twenty-three days after the first operation the hands were separated by dividing the soft-tissue flap which connected the two fingers. Suture of the remaining digital nerve and closure of the wound completed the transplant. *Fig.* 177 show the result eight months after the completion of the second stage.

Case 4.—W. M., aged 12. This boy, who was kindly referred to me by Mr. T. McW. Millar, had most of his right hand blown away by the explosion of an anti-personnel bomb (*Figs.* 178, 179). It will be seen from *Fig.* 179 that the sole remaining bony structures of the hand were the metacarpal and a portion of the proximal phalanx of the middle finger, and the metacarpal of the ring finger. A small range of movement was present at the metacarpophalangeal joint.

This case proved to be extremely difficult and many misfortunes and failures were encountered.

The general aim of reconstruction was the restoration of the structures which subserve the basic function of the hand. It will be recalled that these structures are two digits opposed to each other, at least one of them being mobile. Before this aim was achieved, three separate plans of reconstruction were devised and attempted.

OPERATIVE TECHNIQUE.—

1. *First Plan.*—To construct an artificial digit from living tissues, the digit to be fixed to the anterior aspect of the carpus and thus to face the middle finger; to

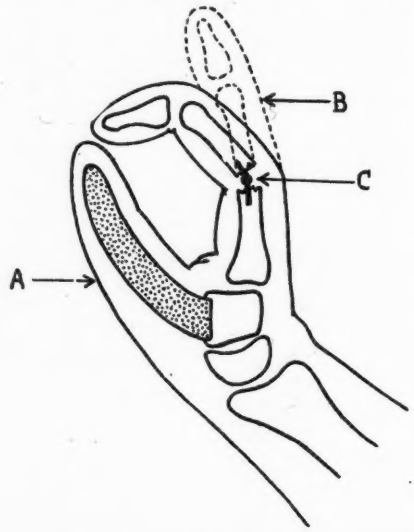


FIG. 180.—Same case. First plan. A, Fixed unit: skin tube and bone-graft. B, Mobile unit: distal half of the metacarpal and proximal phalanx of middle finger. C, Point of action of mobile unit: the metal hinge.

restore mobility to the middle finger; to create an accommodation space between the two digits, the restoration of mobility and the creation of the accommodation space to be achieved by making a false joint in the shaft of the metacarpal (*Fig.* 180).

A tubed pedicle graft was raised from the skin of the right iliac fossa. Through a dorsal incision in the

hand, stainless steel hinges were inserted in the metacarpals after dividing them at the centre of the shaft (*Fig.* 181).

New bone grew round the hinges and firm bony union occurred. A semiflexed position, however, was deliberately maintained. Though the arthroplasty had failed, an accommodation space had been gained by what amounted to an osteotomy (*Fig.* 182). Owing to

FIGS. 181, 182.—Same case. Three weeks after insertion of hinges, showing passive flexion of almost 90°.

the failure to obtain mobility in this digit—the only possible one—the whole plan had to be abandoned.

2. *Second Plan.*—To utilize the carpo-metacarpo-phalangeal unit as the fixed digit and to obtain a mobile digit by a whole finger graft from the uninjured hand (*Fig.* 183). The aim of this plan was: to divide at its centre the metacarpal of the ring finger of the uninjured hand; to bring the distal portion together with flexor and extensor tendons and digital nerves through the

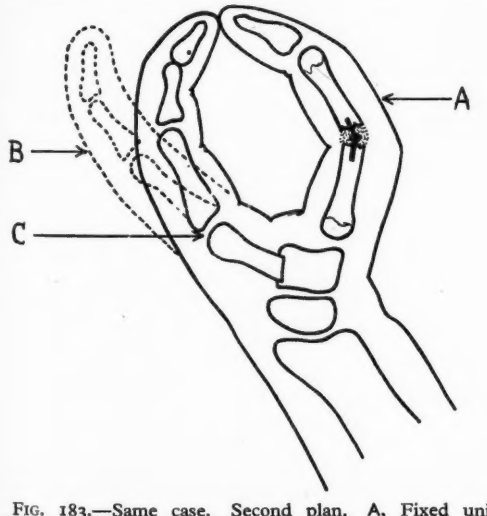


FIG. 183.—Same case. Second plan. A, Fixed unit: metacarpal and proximal phalanx of middle finger. (The stippled area around the hinge indicates bony ankylosis.) B, Mobile unit: all three phalanges of the grafted finger. C, Point of action of mobile unit: metacarpo-phalangeal joint of the grafted finger.

incision into the palm of the hand; then to pass them through an incision on the palmar aspect of the damaged hand, fix the metacarpal to the bones of the carpus, and suture the tendons to the distal ends of similar structures of the injured hand; and finally to complete the union of the two hands by suturing together the corresponding margins of each incision.

Right hand: A palmar incision was made over the carpus. The stump of the median nerve and the tendons of flexor pollicis longus and flexor digitorum sublimis to the index finger were dissected free from scar tissue. A bed was prepared in the carpus for the reception of the left fourth metacarpal.

Left hand: A palmar incision was made over the fourth metacarpal. The digital nerve and artery to each side of the ring finger were sectioned, and the flexor tendon divided high up. A dorsal incision was then made, the extensor tendon divided, and the metacarpal divided obliquely at about its midpoint.

Both hands together: The hands were brought face to face. The distal half of the divided metacarpal was flexed to a right angle at the metacarpo-phalangeal joint and, with the distal portions of the flexor and extensor tendons and digital nerves, brought out through the palmar incision. They were then passed through the palmar incision of the injured hand. The metacarpal stump was fixed to the recipient carpal bed, the flexor digitorum profundus tendon sutured to flexor pollicis longus, and the extensor tendon to the stump of flexor digitorum sublimis to the index finger. The digital nerves were sutured to the end of the median nerve. To complete this stage, the corresponding margins of the palmar incisions in each hand were sutured together, enclosing the above structures and uniting the two hands. The dorsal incision in the left hand was then closed.

At the end of twenty-eight days, a V-shaped incision was made on the dorsum of the left hand, the open ends of the V being in the interdigital clefts adjacent to the ring finger. The incision was deepened till all structures which were to be left behind were reached. Only these structures and the skin and subcutaneous tissues on the palmar side now remained. At this juncture, the circulation of the finger began to deteriorate and the separation was discontinued. The wound was packed open with gauze and the patient returned to bed.

Fourteen days later, the separation was completed by making a similar V-shaped incision on the palmar aspect of the left hand and deepening it until its two limbs met those of the dorsal V. The abdominal skin-tube was utilized at this stage to cover the raw area on the upper and lateral aspects of the grafted finger.

Unfortunately, mild sepsis occurred after the first operation, with the result that tendon action was lost and bony union failed to occur between the donor metacarpal and the recipient carpus. The bone ends were now exposed, freshened, and re-fixed. A free graft of an extensor tendon of the foot was used to replace a portion of the flexor tendon which was irreparably damaged by fibrosis. Again there was a lighting up of sepsis

and again tendon action was lost and bony union failed to occur.

A review of the situation now showed that the actual transplant of the finger had been achieved, but that it

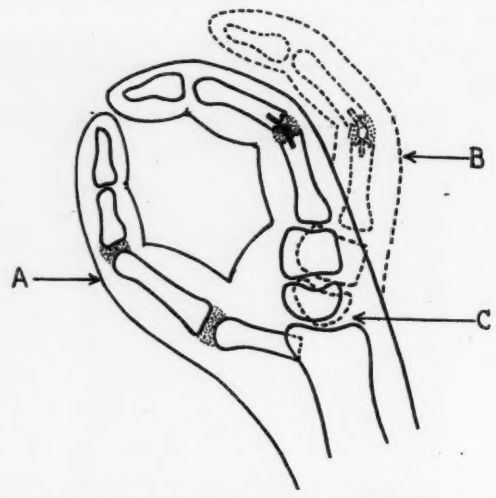


FIG. 184.—Same case. Third plan. A, Fixed unit : whole of grafted finger. B, Mobile unit : carpus, metacarpal, and proximal phalanx of middle finger. C, Point of action of mobile unit : radiocarpal joint. (The stippled areas indicate bony ankylosis.)

was flail and without active movement. With all hope of obtaining active movement now gone, a further attempt to fix the finger to the carpus would have been futile as both digits fixed to the carpus and without independent motion would move together with the carpus. This plan had therefore to be abandoned.

3. *Third Plan.*—To re-graft the finger to the lower end of the radius using it as the fixed digit, the carpo-metacarpo-phalangeal unit acting as the mobile digit (*Fig.* 184).

Through a long antero-lateral incision, the grafted metacarpal and proximal two phalanges were exposed. Both the metacarpophalangeal and proximal interphalangeal joints were excised and a long gutter passing through all three bones on their dorsal aspects was made. A graft was cut from the radius, slid downwards, and locked into the gutter after the whole finger had been moved upwards from the carpus to the distal end of the radius.

At this time penicillin was available and, with its use, sepsis did not occur. *Fig.* 185 show the result of the third plan.

Comment.—W. B. (*Case* 3) is now employed as a bar tender, and his sole complaint is that he is unable to grasp the larger whisky bottles. All other bottles and glasses he handles with ease.

W. M. (*Case* 4) : At the present time, only six weeks after removal of the plaster, there is as yet little power in his grasp, though he is able to hold a cylinder of metal one and a half inches in diameter and weighing one and a half pounds. He can hold a thin sheet of paper and a pin in his finger-tips and can write quite legibly. He is employed as a messenger boy for four hours during the day. At night he attends a technical school, where he is taking a course in engineering.

The response to sensory stimuli in the above two cases is very different. *Case* 3 shows an almost normal response to both exteroceptive and proprioceptive impulses. Pain, temperature, and touch are appreciated in the whole finger with the exception of a small area on the ulnar side of the terminal phalanx. Discrimination is not always accurate, but localization is good and stereognosis is present. Joint position is very accurately determined, and variations of pressure are appreciated. Sweating of the finger occurs, and no trophic changes are present. In *Case* 4 appreciation of both superficial and deep sensibility is absent. The finger does not sweat. It is somewhat tapered and the skin is rather shiny, but ulceration has not occurred. In *Case* 3 tendon and nerve sutures were successfully performed. In *Case* 4 sepsis occurred after the first two operations ; the third operation of regrafting the finger from the carpus to the radius was performed without regard to tendons or nerves.

In the transfer of a finger from one hand to the other, the interval of time between the first and second stages is of vital importance. The loss of a normal finger from the uninjured hand, if due to the too early severance of its pedicle, would be a disaster. In estimating the time when the blood-supply from the recipient hand to the grafted finger is adequate enough to allow of its separation, the tourniquet test may be relied upon. The method of application of this test as used in *Case* 4 is as follows : With the patient in the supine position, the arms with their interlocked hands are raised vertically above the head. Both limbs are then emptied of blood by

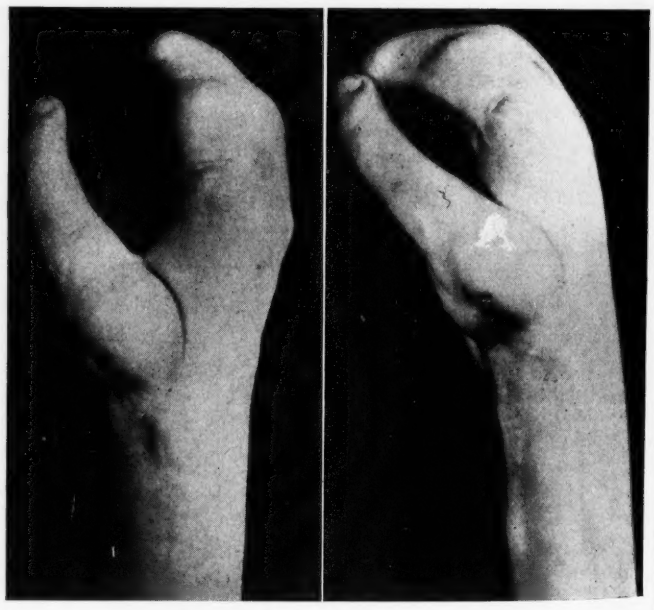


FIG. 185.—Same case. Showing the hand after completion of third plan. Photographs taken immediately after removal of plaster cast.

applying a rubber bandage in an upward direction from the wrist to above the elbow. A second bandage is then applied to each arm as a tourniquet and the first one removed. Both hands are now totally ischæmic and white. The arms are lowered and the tourniquet on the arm of the recipient hand is removed. The flush which follows the tourniquet

removal is watched as it spreads down the limb. If it continues on into the grafted finger, an adequate blood-supply from its host may be assumed and its separation from the donor hand proceeded with.

On general principles, the introduction of foreign material into living tissue should be avoided if possible, particularly in an area where a high degree of function is required, but it is not unreasonable to assume that, with the recent advent of materials which excite very little tissue reaction, in certain cases some form of mechanical hinge could be used as a means of arthroplasty.

A stiff proximal interphalangeal joint is a very crippling disability and presents an extremely difficult therapeutic problem. The results of capsulotomy are very variable and even excision of the joint with the interposition of fat and fascia may fail to give more than a slight improvement in function. In addition, excision often leaves a distorted and unstable finger. In the gross case where the lesser procedure of capsulotomy is unlikely to succeed, a metal hinge arthroplasty perhaps offers the greatest chance of success. In the case quoted above (*Case* 4) it was surprising that bony union occurred when so many of the causes of non-union were present. The bone ends were separated, foreign material was interposed between the fragments, and movement at the fracture was constantly taking place. Active movements were encouraged and passive movements through a maximal range, at first 90° (*Figs.* 181, 182), were carried out daily. Since this operation, a different type of hinge has been used on a number of occasions in both the metacarpophalangeal and the interphalangeal joints, with very encouraging results. (This hinge has a large central portion which occupies the whole of the space between the bone ends.)

The following case serves to justify the operation of hinge arthroplasty. In this patient two adjacent metacarpophalangeal joints presented an equal degree of stiffness, not more than a few degrees of active flexion being present. Both had failed to respond to a capsulotomy owing to the very gross post-infective fibrosis which was present. Both joints were excised in a similar manner. In one, the bone ends were wrapped in fascia and a pad of fat interposed. Into the other a stainless steel hinge was inserted. Both fingers received the same post-operative care and physiotherapy. Exactly the same degree of active flexion was obtained at each joint —nearly 60°. The joint in which excision alone had been performed had a greater degree of lateral instability than the one into which the hinge had been inserted.

Manufacture of an Artificial Digit from Living Tissues

Case 5.—E. C., aged 18. This lad had his right hand crushed in a mechanical hammer. The thumb was lost at the metacarpophalangeal joint, and the middle, ring, and little fingers at the base of the middle phalanges. The index finger, though severely damaged, was saved (*Fig.* 186), but could not be used to reconstruct the thumb as in *Case* 1. It was, therefore, proposed to manufacture a thumb from living tissues. In view of the disadvantages and difficulties associated with a long digit of this nature, it was decided that only a short prop should be used.

Fig. 186.—*Case* 5. Showing loss of thumb and portion of the ulnar three fingers.

Operative Technique.—A tube pedicle graft was raised from the skin of the right flank. In this situation the skin closed readily beneath the tube.

After three weeks, the upper end of the tube was freed and applied over the head of the first metacarpal, after the skin had been excised in the form of an ellipse.

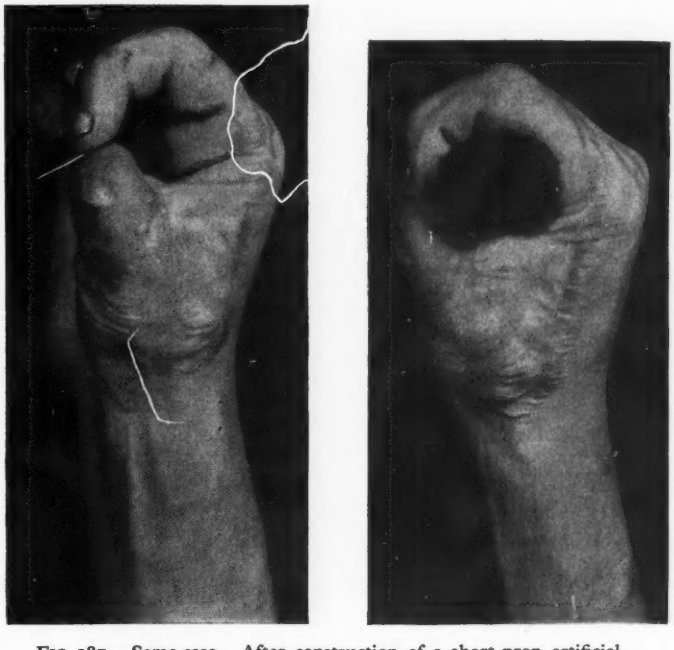


Fig 187.—Same case. After construction of a short-prop artificial digit from a skin tube and free cancellous bone-graft.

In a further three weeks the separation of the tube from the abdomen was completed, and its free end closed by fashioning two flaps of equal length.

Ten months later, a free graft was taken from the iliac crest and implanted in the tube. A curved incision

parallel with the iliac crest and 1 in. below it was made immediately behind the anterior superior iliac spine. The gluteal muscles were stripped from the crest and turned downwards. A portion of crest was then resected with an osteotome, care being taken to leave the cortical bone of the inner aspect of the ilium untouched; the portion excised consisted of the outer three-quarters of the thickness of the crest. The gluteal muscles were then sutured back to the remaining inner portion of the crest. This method of cutting a graft was used to prevent the formation of a hernia which had occurred in an earlier case. The skin tube was opened by a lateral incision and its central core of fibro-fatty tissue excised. The head of the metacarpal was dissected free and a portion of it excised. The free graft was forced into the tube under end-to-end tension, and fixed to the metacarpal by two stainless steel wire sutures passed through drill holes at right angles to each other. *Fig.* 187 show this boy's hand twelve months after operation.

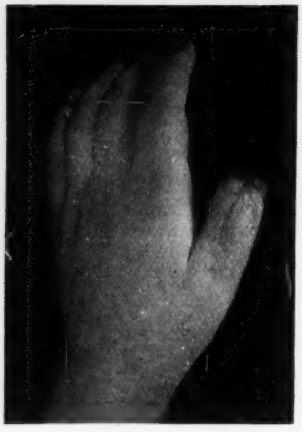
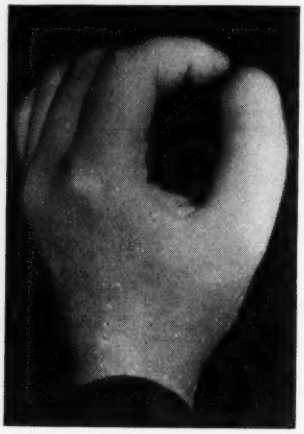
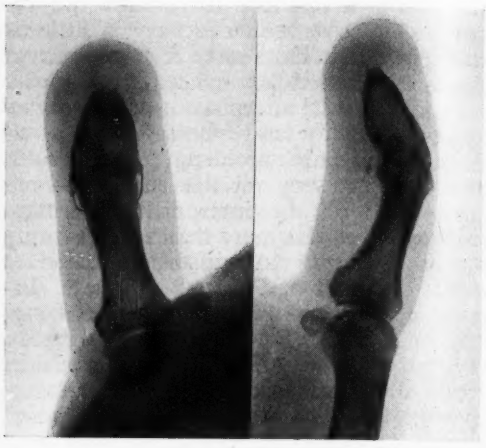


FIG. 188.—*Case* 6. Showing loss of terminal phalanx of left thumb.





FIGS. 189, 190.—Same case. Showing result of replacement of a distal phalanx by a skin tube and free cancellous bone-graft. The radiograph of the bone-graft nine months after operation is similar in appearance to one taken four months after operation.

Case 6.—C. McL., aged 15. This girl lost the terminal phalanx of her left thumb in a bacon-slicing machine (*Fig.* 188). An operation similar in all respects to the one described above (*Case* 5) was performed. *Figs.* 189, 190 show her hand nine months after operation. At the time of the photograph (*Fig.* 190), the thumb was somewhat œdematous, the result of a blow she had received a week previously.

Comment.—In E. C. (*Case* 5), though the new finger lacks length, the function is surprisingly good. Before operation he was unable to pick up small objects and his grasp was weak. Now he is able to hold a pin between the finger-tips, and his grip for anything but large objects is very powerful. The sensory response to touch and pin-prick is accurate. Bony union is very sound and the viability of the finger in general is excellent. During the past eighteen months he has been employed as an apprentice engineer in a machine shop.

C. McL. (*Case* 6) illustrates the use of this method of finger reconstruction from the cosmetic point of view. With the loss of only the distal phalanx of her thumb, function was, of course, little curtailed, but she was very depressed about the appearance of her hand and it was for this reason that the operation was undertaken. Her depression has now left her and she is very grateful for her new thumb. The addition of an artificial nail would be a very great advantage in this type of case. To facilitate the wearing of such a nail, the preparation of a nail fold by overlapping the end flaps of the tube at the time of its removal from the abdomen is at present under consideration.

It has been mentioned that one of the main disadvantages of this method of restoring a lost digit is the lack of mobility. To overcome this disadvantage, the manufacture of a mobile artificial digit from living tissues is, at the moment, under consideration. The general plan of this reconstruction is to insert into the base of the skin tube a free graft of a whole metatarsophalangeal joint from a toe, and when this graft has taken, to insert into the remainder of the tube a cancellous graft using the distal segment of the joint as the new bony host.

SUMMARY

Three methods of restoring lost digits to a hand are described: (1) The transference of a digit of the same hand; (2) A whole finger transplant from the other hand; (3) The manufacture of an artificial digit from living tissues.

Case histories are given in illustration of each method.

The use of a metal hinge as a means of arthroplasty is suggested.

The possibility of constructing a mobile artificial finger from living tissues and of adding an artificial nail to such a digit is mentioned.

I wish to acknowledge my indebtedness to Professor J. R. Learmonth for his encouragement in this type of work and for his helpful criticism and suggestions in the preparation of this paper; to Mr. David Band and other members of the Honorary Staff of Leith Hospital for their co-operation and assistance in establishing a hand unit; to Mrs. Selby Tulloch for the line drawings; and to the nursing staff of Leith Hospital for their patient and careful attention to the many details which these cases involve.

MYELOGRAPHY IN LUMBAR INTERVERTEBRAL DISK LESIONS

A CORRELATION WITH OPERATIVE FINDINGS

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The value of myelography in the investigation of patients thought to be suffering from intraspinal protrusion of the lower lumbar intervertebral disks is still a matter of controversy. At first, when this new field of surgery was being explored, an abnormal myelogram was considered essential for diagnosis (Mixter and Barr, 1934; Hampton and Robinson, 1936; Love and Camp, 1937), but with increasing experience it was found that the diagnosis could be made readily from clinical features alone, and, indeed, even more accurately, for some disk prolapses could not be demonstrated by myelography (Semmes, 1939; Bradford and Spurling, 1941; McKenzie and Botterell, 1942). It was also learnt that the myelographic appearances might be difficult to interpret, and could be confusing or misleading, and this prompted some authorities to discredit the procedure (Dandy, 1941 and 1943; Skinners and Hamby, 1944). More recently, however, other investigators employing pantopaque as the contrast medium have claimed that they can demonstrate lumbar disk protrusions with complete accuracy (Childe, 1945; Soule, Gross, and Irving, 1945), while yet others using the same medium have found that the myelogram may be normal, and yet a disk protrusion be present (Eaglesham, 1944; Arbuckle, Shelden, and Pudenz, 1945).

This controversy appears to be the result of an incomplete appreciation of the uses and limitations of myelography in intervertebral disk protrusions. We have therefore analysed our experiences in 100 consecutive cases of lumbar disk lesions submitted to operation. In this series the diagnosis was based on history and physical signs alone (Falconer, 1944). None the less, myelography was employed as a routine, for we found that this procedure is valuable in revealing not only the level of a disk protrusion, but also its site in relation to the theca and extrathecal nerve-roots. Thus the myelogram shows whether a protrusion is centrally placed or laterally placed—information which is helpful, for at operation it has often proved necessary to deal with a centrally placed prolapse from both sides of the theca, rather than by a unilateral approach. Further, multiple disk protrusions are not infrequently present, a fact which it is also useful to know before operation. Finally, myelography may disclose cauda equina tumours and other lesions which simulate an intervertebral disk syndrome.

Thus myelography tells the surgeon beforehand what to expect at operation. As a surgeon forewarned is a surgeon forearmed, a consideration of our experiences may assist others.

NOMENCLATURE

In the literature a number of terms have been used to describe intraspinal protrusions of intervertebral disk tissue. Among the terms which have become almost synonymous through usage are prolapse, protrusion, rupture, and herniation. Because of this confusion of terminology, it seems desirable to define at the outset the terms which we have found useful in describing different types of lesion. In common with others we employ 'prolapse' and 'protrusion' as synonymous generic terms covering all types of these intraspinal lesions. From our operative and myelographic experience, however, we feel that these lesions may be subdivided with advantage into four separate groups, as follows:—

1. *Projections.*—The commonest type is a constant, localized projection of nuclear substance still covered by the smooth, glistening, annulus fibrosus, thinned but intact.

2. *Intermittent Prolapses.*—Next in frequency is an intermittent prolapse which may protrude only when the intervertebral disk is subjected to certain strains. This type is probably similar to the 'concealed' disk described by Dandy (1941).

3. *Extrusions.*—In some cases the annulus fibrosus ruptures, allowing nuclear material to escape free into the spinal canal. This we term an 'extrusion'.

4. *Scarred Disks.*—Over a period of years the opposing surfaces of the vertebral bodies may become sclerotic, the disk space may become greatly diminished in width, and at operation the theca and extrathecal nerve-root may be found firmly adherent to the back of the disk without an actual projection being present. This we call a 'scarred disk'.

CASE MATERIAL

Myelography, employing either 'Pantopaque' or 'Lipiodol Lafay' as the contrast medium, was carried out in 95 of 100 consecutive cases of sciatica and low back pain occurring singly or in combination, while in 1 case an oxygen myelogram was used. The clinical features, operative findings, and results of treatment of this group will form the subject of another study. With one exception disk lesions were found at operation in all cases. Some patients were explored more than once because the pathological disk tissue had been inadequately removed at the first operation. Eventually all cases of sciatica were benefited, as were the majority of cases of severe low back pain unaccompanied by sciatica. During the investigation we encountered one case of tumour causing sciatica. This was an extrathecal hydatid cyst which was distinguishable by myelography. It would seem from our experience that intervertebral disk lesions are the only common cause both of sciatica and of severe low back pain.

Early in the series we encountered 9 cases in which the myelograms were worthless because of artefacts arising from faulty technique. By experience we learnt the causes and the significance of these artefacts, and later how to obviate them. The principal analysis is based on the 86 cases in which myelograms

were satisfactory. In 42 of these pantopaque was the contrast medium, and in the remainder (44) lipiodol. Pantopaque was employed for preference, but as supplies were not always available, lipiodol had at times to be used instead. Myelography was not carried out in 4 cases because no suitable medium was available at the time.

METHOD OF EXAMINATION

Our method of examination resembles that employed by most authorities, but it differs in certain respects. We inject 3 c.c. of contrast medium through a lumbar-puncture needle inserted at the third or fourth lumbar interspace with the patient lying on his side. The needle is then withdrawn. The examination is carried out as soon as convenient, the patient meanwhile remaining recumbent.

The examination is begun with the tilting table vertical, and the patient standing on the footplate facing it. Thus, at the outset, the contrast medium is collected in a mass in the thecal cul-de-sac. Then, as the head of the table is lowered, it flows evenly up the lumbar canal, its progress being watched carefully on the fluorescent screen. Most patients need not move during the whole examination, but occasionally it may be necessary to rotate a patient into an oblique position to obtain better filling of the root pouches, or to investigate further a suspicious filling defect. As the medium straddles each disk space, anteroposterior films are exposed with the spot film device, while the angle of tilt of the table is noted. Once all the lumbar interspaces have been inspected, tilting of the table is reversed, and the opaque medium observed as it flows slowly back to the bottom of the lumbar sac. The constancy of an abnormality may thus be established, and, if necessary, the whole manœuvre may be repeated. Prone-lateral views of the lower two lumbar interspaces are taken as a routine, and this is facilitated by placing the tilting table at the angles previously noted. An independent rotating-anode tube and a wafer grid are used. We have found these lateral views most helpful in the subsequent interpretation, although many authorities have apparently found them of little use (Hampton and Robinson, 1936; Childe, 1945; Soule, Gross, and Irving, 1945). Oblique views, on the other hand, rarely reveal information which cannot be derived from a study of the anteroposterior and lateral projections.

Recently we have introduced a manœuvre which we believe increases the scope of the examination, especially in cases where the myelograms at first sight seem normal. While the opaque medium lies in relation to each disk space, hyperextension of the spine is effected by asking the patient to raise his head and shoulders as far off the table as possible, without altering the position of the lower portion of the trunk. In this way we have been able to demonstrate lesions which the usual technique had failed to disclose. These lesions are of the type which we describe as 'intermittent prolapses'.

In only a few cases was the contrast medium subsequently removed by aspiration through a lumbar-puncture needle as recommended by Kubik and Hampton (1941). Lipiodol we generally aspirated at operation after the theca had been opened. Pantopaque, however, was often left in situ, and no harmful effects have been noticed. This has enabled us to screen our patients at intervals after the initial myelogram. Filling of the root pouches may not be complete for some days, and so delayed myelography has occasionally demonstrated abnormalities which were not visible at the first examination. Important information regarding the persistence of defects has also been obtained in patients whose symptoms had disappeared spontaneously. Follow-up radiological studies have confirmed that pantopaque is slowly absorbed, at a rate approximately two-thirds of its volume in a year.

RELEVANT ANATOMICAL CONSIDERATIONS

The myelographic appearances depend upon the shape of the subarachnoid space, and the extent to which it is filled by the contrast medium. They are also influenced by structures within the subarachnoid space or in proximity to it. Consequently a consideration of the relevant anatomical relationships is important, particularly as in the literature some terms are employed confusingly.

By the term theca we refer to the meningeal tube composed of dura and arachnoid which surrounds the spinal cord and cauda equina. This tube presents a number of prolongations or sheaths which

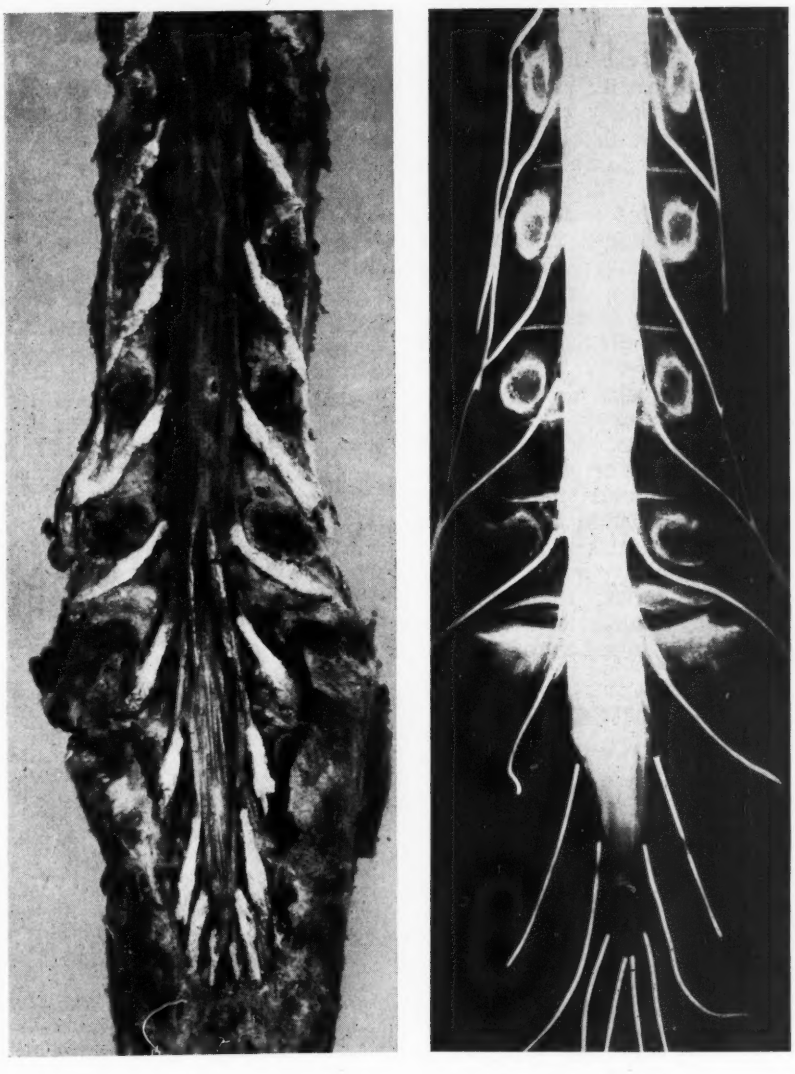
FIG. 191.—Diagram of a root pouch illustrating relationship of dural and arachnoidal sheaths to a nerve-root in its extrathecal course. The subarachnoid space is coloured pink, the arachnoid is depicted by a thin line, the dura by a thick line, the intervertebral disc by cross-hatching, and the pedicle by the number of its vertebra.

surround emerging nerve-roots in the first part of their extrathecal course. Each sheath is composed of two layers: the outer, formed by dura, passes on to become the neurilemma of the spinal nerve; the inner, derived from the arachnoid, forms a sleeve which invests the nerve-root for a variable distance, and may reach as far as the dorsal root ganglion before it fuses with the root pia. Hence a prolongation of the subarachnoid space accompanies each nerve-root, and this prolongation constitutes

the *root pouch* (*Fig.* 191). The root pouches of the first and second sacral nerves are the longest.

The elements of the cauda equina lie loosely within the subarachnoid space, where they are freely movable. Each nerve-root consists of a dorsal and a ventral component in close apposition. As it proceeds towards its point of emergence from the theca, it passes laterally and forwards to lie in contact with the antero-lateral wall of the theca for some distance. On entering the root pouch it becomes relatively fixed, and therefore from this point onwards it is more readily affected by external pressure. From the theca it proceeds downwards, outwards, and slightly forwards, around the infero-medial aspect of the vertebral pedicle to the inter-vertebral foramen where its spinal (dorsal root) ganglion is situated.

The arrangements of the individual nerve-roots composing the cauda equina usually conform to one of two main patterns, but intermediate variations occur. These patterns may influence the myelographic appearances within the theca (*see Fig.* 193).



A B

FIG. 192.—A, Dissection of the lumbosacral region from behind. The pedicles had been sawn through, the laminæ removed, and the thecal sac and extrathecal nerve-roots displayed. The nerve-roots in their extrathecal course have been painted white. B, Radiograph of this dissection after lipiodol had been introduced into the subarachnoid space and wires placed in positions corresponding with the extrathecal course of the nerve-roots. The radiograph shows clearly the relation of the nerve-roots to the root pouches, and also to the intervertebral discs. (× ⅜.)

In one pattern the individual nerve-roots are separated from each other, and are so spaced that those about to pass through the theca lie laterally, with the others lying in order on their medial aspect. The filum terminale lies between the lowest nerve-roots of either side. In the other pattern the nerve-roots are arranged in two large bundles, one on either side of the midline, with a wide free space between. Within each bundle the nerve-roots are loosely adherent to one another, and are so placed that each lies posterior or postero-medial to its next cephalic neighbour. The filum terminale may lie by itself

in the interval between the two bundles, or it may be attached to the medial margin of one or other bundle.

The anterior wall of the theca is closely applied to the posterior surfaces of the intervertebral disks, being separated from them only by the posterior longitudinal ligament. If, however, the lumbosacral angle is more acute than usual, the theca may be separated a few millimetres by fatty tissue from the fifth lumbar disk. This explains why disk protrusions at the fourth and higher lumbar spaces affect the myelograms more readily than do protrusions at the fifth space. The first sacral nerve-roots emerge from the theca at or just above the level of the fifth lumbar disk. The fifth lumbar nerve roots emerge at or just below the fourth lumbar disk, while nerve-roots higher up all emerge below the disk cephalic to their numerically corresponding vertebra (*Figs.* 192, 197). Consequently disk protrusions situated posterolaterally at the fourth and fifth lumbar spaces readily compress the fifth lumbar and first sacral nerves respectively, while similar protrusions at higher levels usually do not involve an extrathecal nerve-root.

A difficulty in the enumeration of lumbar disk spaces arises when there are more or fewer than five lumbar vertebræ. We have found then that the distribution of nerve-roots in relation to the lowest or lumbosacral disk usually indicates that it corresponds to the fifth lumbar disk of persons with five lumbar vertebræ. In this series, therefore, we have numbered the lowest lumbar disk space as the fifth, irrespective of the number of lumbar vertebræ present.

THE HYDRODYNAMICS OF MYELOGRAPHY

The principle underlying myelography is to introduce into the spinal subarachnoid space some innocuous medium which will outline it when examined by X rays. There are, however, certain points about the hydrodynamics of myelography which, though simple, are not widely appreciated, and which must be understood if myelograms are to be interpreted properly.

The two contrast media, pantopaque and lipiodol, are both iodized oils heavier than cerebrospinal fluid and not miscible with it. Consequently, when int oduced into the theca in limited amounts, each substance gravitates to the most dependent portion of the subarachnoid space, where it forms a pool with a horizontal upper surface, above which is cerebrospinal fluid. The shape, depth, and location of this pool alters with the position of the patient. If he stands erect, the pool collects in the sacral cul-de-sac, where its cross-section is limited by the circumference of the theca, and consequently its depth is relatively great. But if the patient lies horizontal in the prone position, the medium will flow towards and cover the anterior wall of the lumbar theca, for that is now the most dependent portion of the subarachnoid space. There it will again form a pool with cerebrospinal fluid intervening between its upper surface and the dorsal wall of the theca. This time, however, the longitudinal expanse of the pool is great, because the lumbar theca is now also horizontal, while its depth is shallow in roughly inverse proportion to its length.

If sufficiently large quantities are introduced, the medium will fill the whole cross-section of the lumbar theca, even when the patient lies horizontally. But we believe there are disadvantages in this, and that 3 c.c. is the optimum amount of any oily contrast medium which should be introduced. This amount when collected along the anterior wall of the lumbar theca forms a pool which is usually sufficient to straddle one disk space at a time, but not two. The pool is relatively shallow, and consequently small projections will

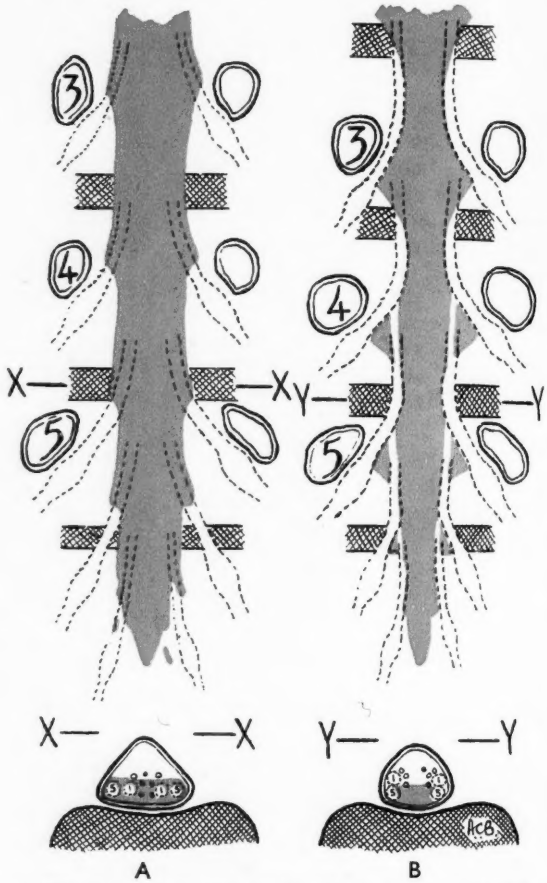


FIG. 193.—Composite tracing of an actual myelogram depicting: A, The 'cylindrical' type; and B, The 'ringed' type of thecal column. Below each tracing is a schematic cross-section of theca at level of 4th lumbar disk, showing nerve-roots with contrast medium in relation to them. Note the two different patterns of the cauda equina.

show through it. If less than 3 c.c. is employed, there may be insufficient medium to outline adequately that portion of the theca which is in relation to the intervertebral disk, especially when the theca is broad. If more than 3 c.c. is used, the pool may be so deep that small projections cannot be seen through it, as its fluoroscopic shadow is too dense. Furthermore, these larger amounts may cover the fifth disk even when the patient stands erect, thus preventing a complete examination of this space.

One disadvantage of both these oily contrast media is their tendency to break up into globules. Lipiodol is a greater offender in this respect than is pantopaque. If on lumbar puncture these substances are injected slowly and steadily, they will usually

collect into one uniform mass. Thereafter care should be taken that the patient is tilted slowly so that the medium flows in one mass, otherwise portions will break off and separate into globules. Once fragmentation has occurred, it may be impossible to fuse the contrast medium together again.

THE NORMAL MYELOGRAM

It is convenient to consider the silhouette of a normal myelogram under two headings, the thecal column and the root pouches.

1. Thecal Column.—A striking and constant feature of the normal thecal column as seen in the anteroposterior view is its symmetry. Variations occur in its shape, width, and length. There are two main types of column, a cylindrical column of even tubular form, and much less commonly a ringed column with zones of constriction between the levels at which successive pairs of nerve-roots emerge. All intermediate grades are seen between the extremes depicted in *Fig.* 193. When the pool of contrast medium is deep, the thecal shadow is of uniform density. When, however, the pool is shallow, the nerve-roots show through as bands of

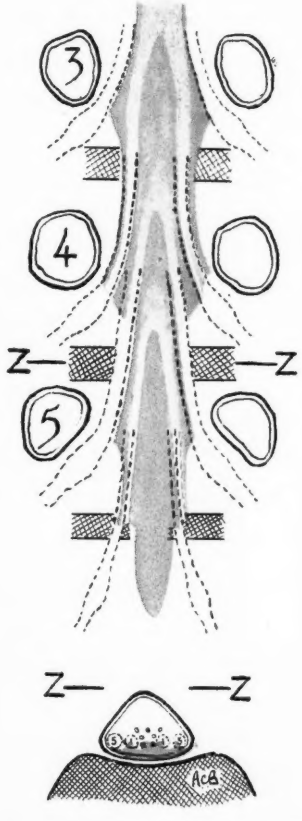


FIG. 194.—Composite tracing of actual myelogram indicating the topographical arrangement of the cauda equina.

radiolucence, particularly if they lie in close apposition to the anterior wall of the theca. This latter appearance is likely to occur in patients with a wide theca and a relatively straight lumbar spine. It may even be possible to appreciate the arrangement of the cauda equina (*Fig.* 194).

The width of the thecal column may vary from 30 to 80 per cent of the interpedicular distance (*Fig.* 195), while its depth, too, may vary. These variations are not directly related to the sex or build of the patient, for we have seen narrow columns in

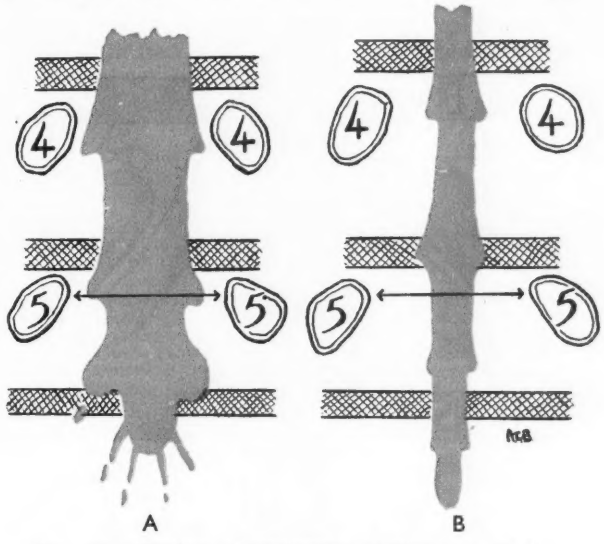


FIG. 195.—Tracings of myelograms showing: A, Broad type; and B, Narrow type of thecal columns.

heavily built men, and broad columns in young, slender women.

Generally the thecal column extends as far down as the second piece of the sacrum, but occasionally it may terminate as high as the inferior margin of the fifth lumbar vertebra. When this occurs the root pouches of the sacral nerves are often large and fill well, and consequently the effects of a protrusion of the lumbosacral disk may still be appreciated (*Fig.* 196).

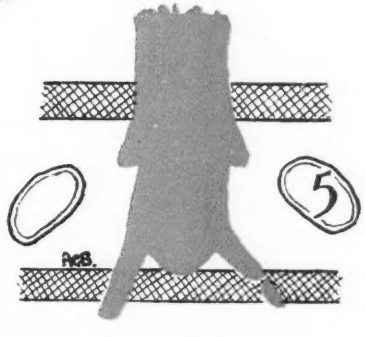


FIG. 196.—Tracing of myelogram, showing high termination of thecal sac.

Prone-lateral views show that, posterior to each intervertebral disk, the anterior wall of the theca is normally indented by the disk and its ligamentous coverings to a depth of up to 2 mm. These small indentations are of no pathological significance, but they may cause an exaggerated and misleading deformity in the upper lumbar region where the spine is straight and the pool of contrast medium consequently shallow. On the other hand, at the fifth space where the theca may not be in contact with the intervertebral disk, an anterior indentation is often absent.

2. Root Pouches.—The outlines of the root pouches also are usually symmetrical, but the degree to which they fill varies considerably (*Fig.* 197). Certain factors affect the filling, of which time, gravity, and the posture of the patient are the most

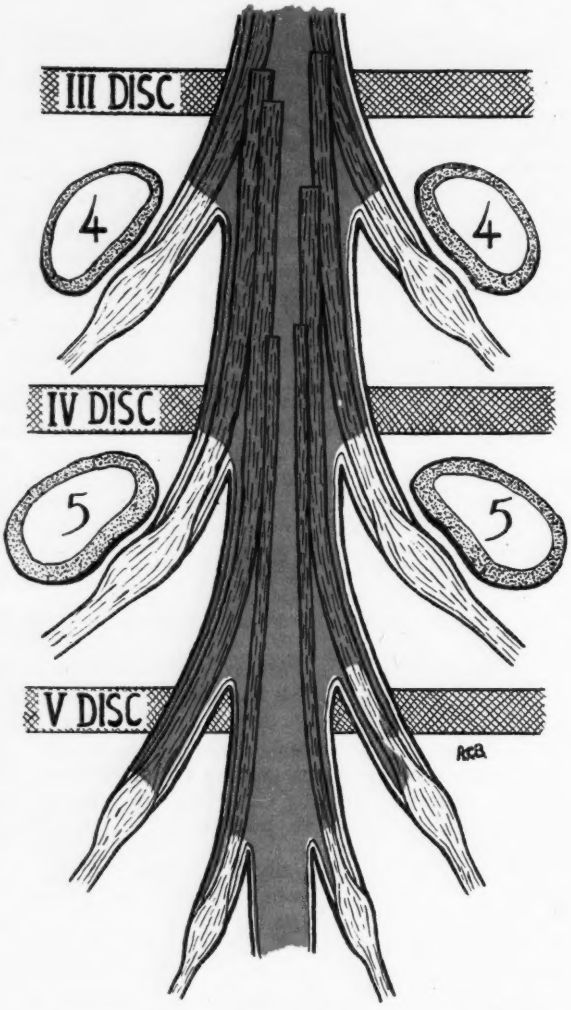


FIG. 197.—Diagram of a normal myelogram in the lumbo-sacral region, showing the variations which may occur in the filling of the root pouches with contrast medium, represented by pink wash.

important. Thus, if the radiological examination is made immediately after the injection of contrast medium only the orifices of the root pouches may be demonstrated, but if the examination is repeated some days later the sacral root pouches are usually filled in their entirety. The lumbar root pouches, however, do not fill as completely as the sacral pouches, because the contrast medium does not remain in relation to them when the patient sits, stands, or lies.

When filled completely, each root pouch shows as a uniformly dense shadow 3 to 4 mm. wide passing downwards and outwards from the main thecal column. If the layer of contrast medium between the nerve-root and the arachnoid is thin, the root pouch may show in the myelogram as two linear streaks with a clear zone between, representing the nerve-root. More frequently, where filling is irregular, the opaque medium may appear as a single streak lying medial or lateral to the nerve-root. The first and second sacral pouches are the longest and may measure as much as 3 cm. in length, while the root pouches higher up are progressively shorter. Often, however, the root pouches do not fill with contrast medium, in which case their orifices are indicated by small projections along the lateral edge of the thecal column (*Fig.* 197).

THE PATHOLOGICAL MYELOGRAM

Prolapse of a lumbar disk usually affects the thecal column or a root pouch. Occasionally, the myelogram may be normal. We have been able to correlate the myelographic appearances with the findings at operation, both as regards the level and the size of the prolapse, as well as the type of lesion.

A. LEVEL AND SITE OF PROLAPSE

Usually the myelographic abnormalities are situated opposite a disk space, thus indicating the level of the lesion. The site of the prolapse in relation to the theca and extrathecal nerve-roots may also be determined with accuracy. Disk prolapses may be divided into two groups, central and lateral, according to their location in the coronal plane. Central prolapses lie anterior to the theca, while lateral protrusions lie between them and the intervertebral foramen (*Fig.* 198). Lesions in either of these situations have their own characteristic appearances on myelography.

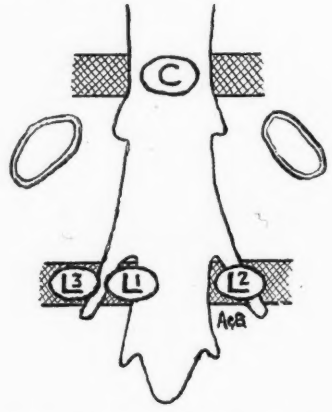


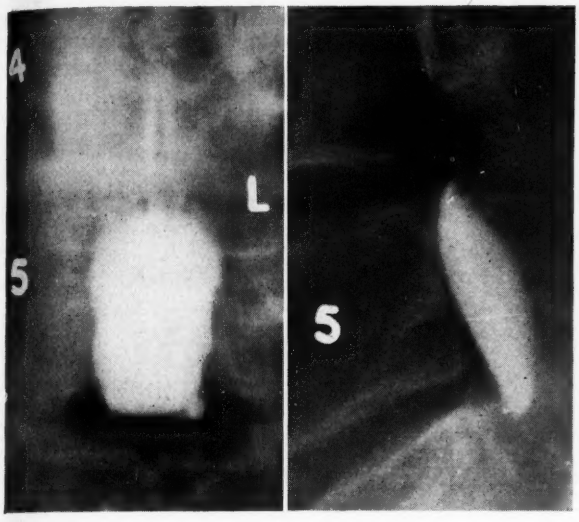
FIG. 198.—Diagram illustrating sites of central and lateral prolapses.

1. Central Prolapses.—The appearance caused by a central prolapse depends mainly upon the size of the prolapse, but it is also influenced by the arrangement of the cauda equina, and by the variations in distance occurring between the theca and the intervertebral disks at different levels of the spine. If the prolapse is large enough, a complete block will result accompanied by Froin's syndrome (*Fig.* 199). It is, however, exceptional for a block to remain permanent and absolute, for if the examination is repeated a few hours later some of the contrast medium will be found to have passed the obstruction, although during screening it could not be persuaded to do so.

Complete blocks are uncommon, for they occur only with the largest prolapses. With medium

sized prolapses the usual finding is an hour-glass constriction of the thecal column, the width of the isthmus varying considerably, as is shown in *Fig.* 200, A, B. In the past this 'hour-glass' appearance has been ascribed either to hypertrophy of the ligamentum flavum (Hampton and Robinson, 1936; Camp, 1939) or to a bilateral or dumb-bell-shaped prolapse (Soule, Gross, and Irving, 1945). We cannot, however, confirm either of these explanations, for in our experience it is due to a smooth, dome-like protrusion, which invaginates the anterior wall of the theca in the midline and displaces the components of the cauda equina into two bundles, one on either side. Where the two bundles pass over the prolapse, they hug closely the anterolateral wall of the theca, leaving a gutter of varying width between them, through which the contrast medium flows (*Fig.* 201). Prone-lateral views of these 'hour-glass' constrictions have invariably disclosed an anterior invagination of the thecal column (*Fig.* 200, C) and operative exploration has later confirmed it. The 'gap' appearance shown in *Fig.* 202 is only an early stage in the filling of the gutter, although it has been described by some authorities as a separate type (Soule, Gross, and Irving, 1945). On screening, the contrast medium, once it has reached the prolapse, can be seen to bank up and spill over it, and then lie on either side of it. When with further tilting of the table the pool deepens, an hour-glass appearance becomes evident.

A less common appearance with a medium-sized prolapse is a rounded zone of radiolucence lying within the thecal column and causing no deformity of its margins as seen in the anteroposterior view. This appearance, the 'veil' effect of Soule, Gross, and Irving (1945), results when the components of the cauda equina lie discretely over the summit of the ridge, instead of in two large bundles. On screening, the advancing column can be seen to split and flow round either side of the projection, although later, as the pool deepens, the projection becomes



A B

FIG. 199.—Large central prolapse of L.4 disk causing complete block, with contrast medium trapped caudal to it. Films taken with table tilted head down 60° below horizontal. A, Anteroposterior view showing serrations at edge of block due to nerve impressions; B, Lateral view, showing that cephalic end of column is displaced backwards by the prolapse. (× ⅔.)

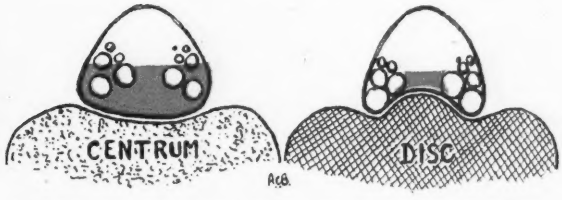
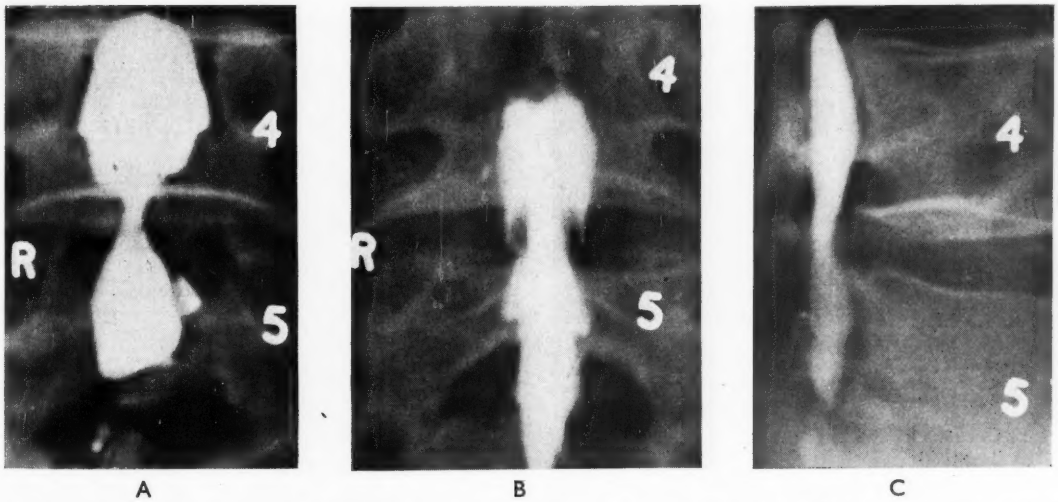


FIG. 201.—Diagrams showing how the 'hour-glass' appearance is produced. A, Cross-section opposite vertebral body shows undisturbed position of nerve-roots within contrast medium (pink wash). B, Cross-section at level of disk, showing contrast medium lying over summit of midline protrusion in gutter between bundles of nerve-roots.

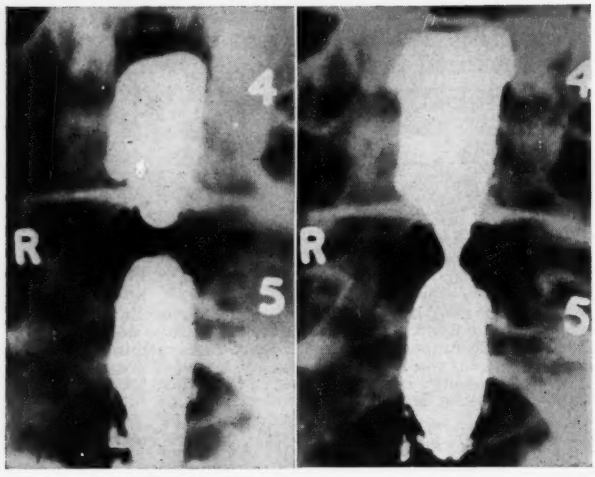


A B C

FIG. 200.—Two examples of hour-glass constriction of the thecal column, showing variations in width of the isthmus: A, Shows a narrow gutter. B, Shows a wider gutter. Note the outlines of nerve-roots as they hug the anterolateral wall where stretched over the dome of the prolapse. C, Prone-lateral view of lesion depicted in A, showing the anterior invagination of the thecal column. (× ⅔.)

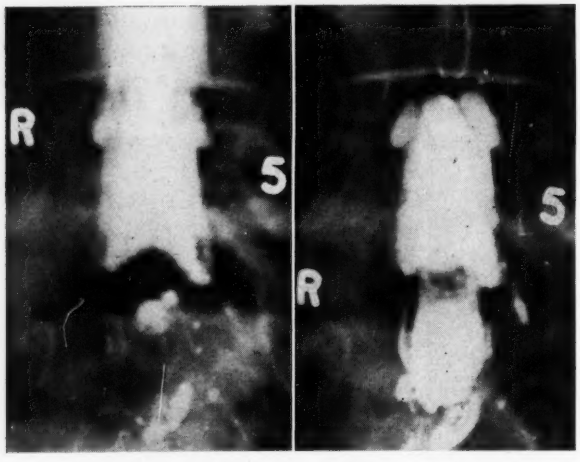
submerged and finally obscured (*Fig.* 203). Copleman (1944) has made similar observations.

In smaller-sized protrusions, and even in some of the larger protrusions at the fifth lumbar space, the anteroposterior views may appear normal, and the anterior invagination of the theca be detected only in the prone-lateral projection. This is another reason why we feel that prone-lateral views should be taken as a routine. Normally, there is a slight indentation of the theca opposite each disk space, but these indentations are only of pathological significance when they exceed about 2 mm. Another source of difficulty arises with centrally placed prolapses at the fifth lumbar disk, where, because the theca may here be separated from the intervertebral disk, a prolapse of given size may produce less indentation than a comparable prolapse at a higher level. This explains why the 'hour-glass' appearance is seen much more often at the fourth lumbar space than at the fifth. The size of all midline protrusions is best assessed in the prone-lateral view.



A B

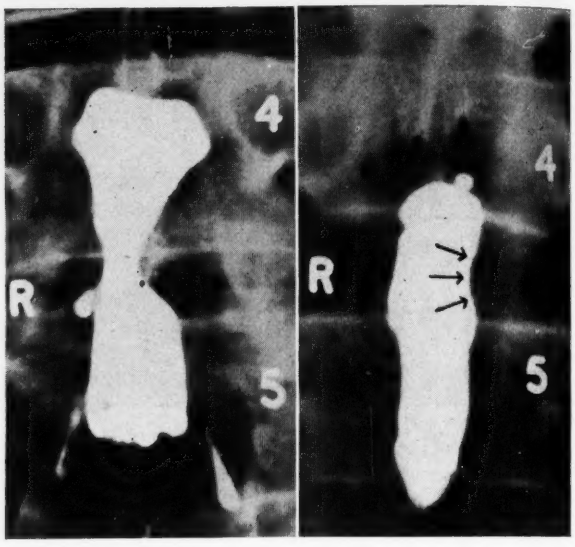
FIG. 202.—Illustrates two successive stages in the passage of contrast medium over a central prolapse. A, There is an apparent gap in the column; while in (B), the medium is seen flowing through the gutter over the summit of the prolapse. (× ⅔.)



A B

FIG. 203.—An example of 'veil' appearance due to a centrally-placed prolapse. A, The advancing column diverges on either side of the prolapse; B, The prolapse is revealed by a central area of radiolucence. (× ⅔.)

2. Lateral Prolapses.—When a prolapse occurs in relation to an extrathecal nerve-root, the myelographic appearance varies according to the distance the prolapse is situated from the mid-sagittal plane. Lesions situated anteromedially to the extrathecal nerve-root (*see Fig.* 198—L.1) give rise to a defect in the lateral border of the thecal column, while the corresponding root pouch usually fails to fill (*Fig.* 204). The size of this defect varies and does not indicate accurately the size of the prolapse, as only its medial margin is outlined. A prone-lateral view sometimes reveals the size of the prolapse by a



A B

FIG. 204.—Two examples of lateral prolapse indicated by a defect in the thecal column: A, The defect is large, indicating a large prolapse; B, The defect is small, but this does not necessarily mean the prolapse is small. (× ⅔.)

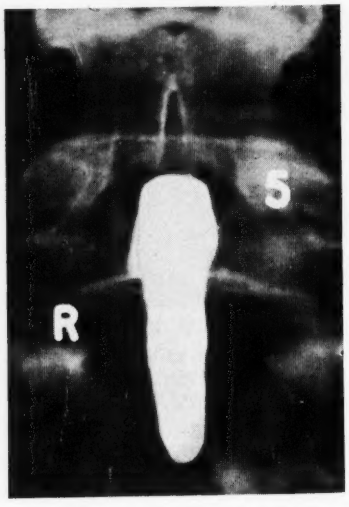


FIG. 205.—Lateral prolapse indicated by absence of filling of S.1 root pouch opposite 5th disk on left side. (× ⅔.)

double shadow. If the prolapse is small it is best seen as the advancing column reaches the disk space or as the receding column leaves it, since it may be completely obscured by the main mass of the opaque column.

Lesions situated anterior or slightly antero-lateral to the nerve-root (*see Fig.* 198—L.2) cause a defect in the filling of the root pouch without disturbing the thecal column (*Fig.* 205). We have found that the deformity may be due either to direct pressure of the prolapse on the root pouch or to swelling of the nerve-roots within the pouch proximal to the site of compression.

FIG. 206.—Large prolapse situated far laterally at 5th lumbar space on right side, producing displacement of thecal column by its pressure on 1st sacral nerve-root. (× ⅔.)

When the prolapse is situated farther laterally (*Fig.* 198—L.3) the myelogram may show a characteristic change or it may remain normal. If the lesion is a large one its presence may be shown by displacement of the nerve-root, and indirectly of the whole thecal column, towards the contralateral side (*Fig.* 206). If the myelogram remains normal in the presence of strongly suggestive clinical features, we have found that a prolapse is likely to be situated in the foraminal region at the fifth space (*Fig.* 207).

B. TYPES OF PROLAPSE

As mentioned earlier, we have subdivided our cases of prolapse into four groups, projections, intermittent protrusions, extrusions, and scarred disks. This classification is based on operative appearances, which by means of myelography can sometimes be forecast.

1. **Projections.**—The appearances which have so far been described are those caused by projections.

2. **Intermittent Prolapses.**—All who have had wide experience of myelography know that, although the silhouette of a disk prolapse may have been demonstrated radiologically, the surgeon sometimes will be unable to see a projection when he examines the disk at operation. Less frequently the converse occurs, and the surgeon operating despite a normal myelogram confronts the radiologist with a well-marked prolapse; some of these latter prove to be examples of foraminal projections at the fifth lumbar disk remote from the theca and root pouches, but not always.

Observations such as these have in the past brought discredit upon myelography. It has seldom been realized that the projection of a prolapse may vary markedly according to the stresses which are being imposed on the affected disk. Dandy (1941), for instance, stressed that at operation the affected disk may scarcely bulge, and when palpated with the points of forceps it may indent and give a sense of fluctuation. Disk prolapses of this type he referred to as 'concealed' disks, and he assumed that, as the degree of projection of these disks seemed so slight, they would not be disclosed by myelography.

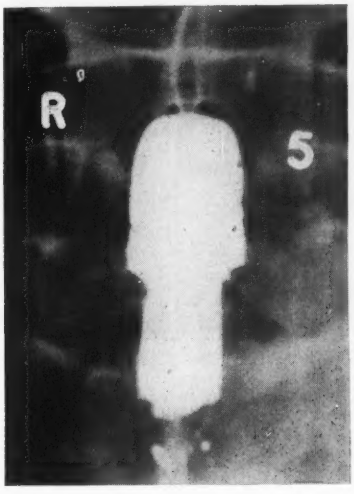


FIG. 207.—Normal myelogram of a patient in whom a lateral projection was found at operation at the 5th space on the left side. (× ⅔.)

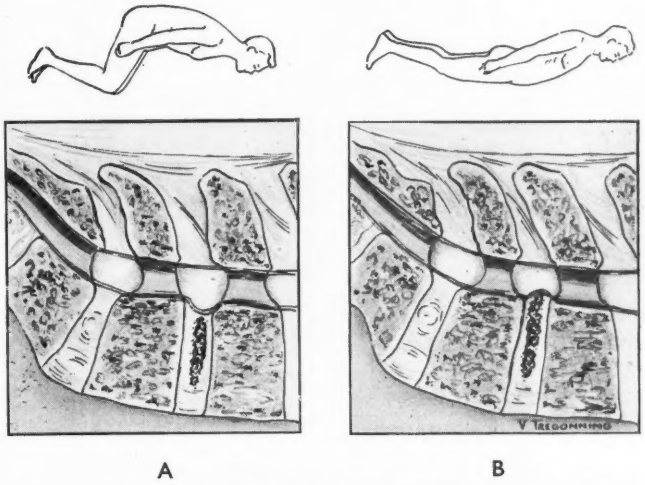


FIG. 208.—Diagrams showing effect of 'hyperextension' manœuvre as performed at operation. A, There is no projection backwards of the weakened disk, which is relaxed; B, With hyperextension the disk protrudes backwards.

Few radiologists and surgeons appear to have realized that there is a great difference in the stresses and strains affecting the lumbar disks of a patient on

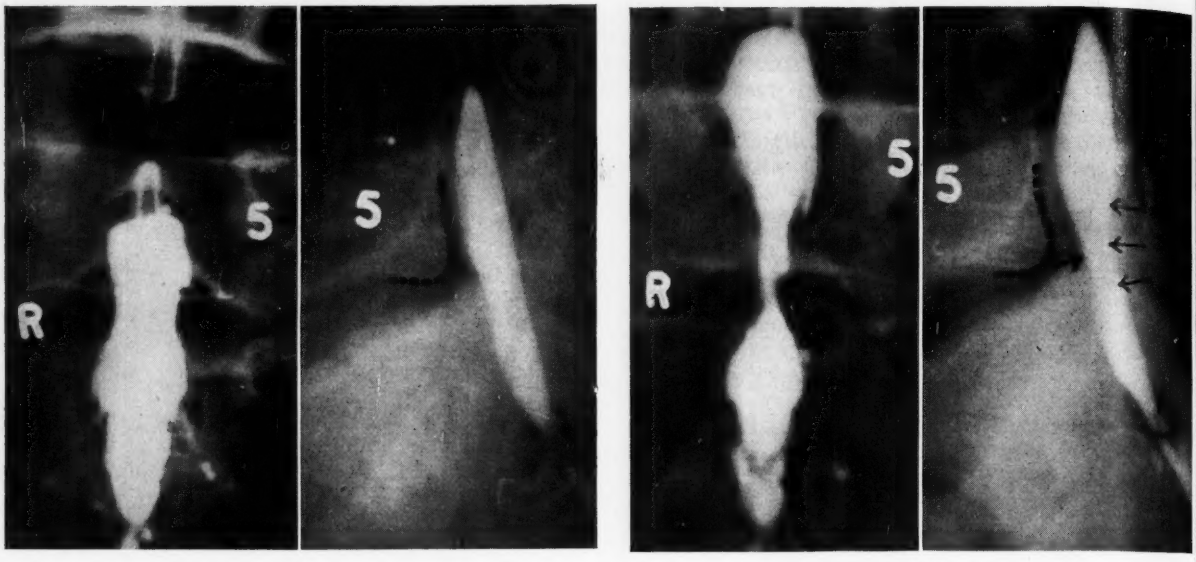


FIG. 209.—Myelograms showing large intermittent central prolapse at lumbosacral space revealed by ' hyperextension ' manœuvre. A, B, Routine myelogram with patient relaxed shows no abnormality in anteroposterior or prone-lateral views; C, D, Myelogram during ' hyperextension ' manœuvre demonstrates the presence of a prolapse anteriorly, as well as the impression of a relaxed ligamentum flavum posteriorly.
The postero-inferior outline of the 5th lumbar vertebra is marked by a dotted line. (× ⅔.)

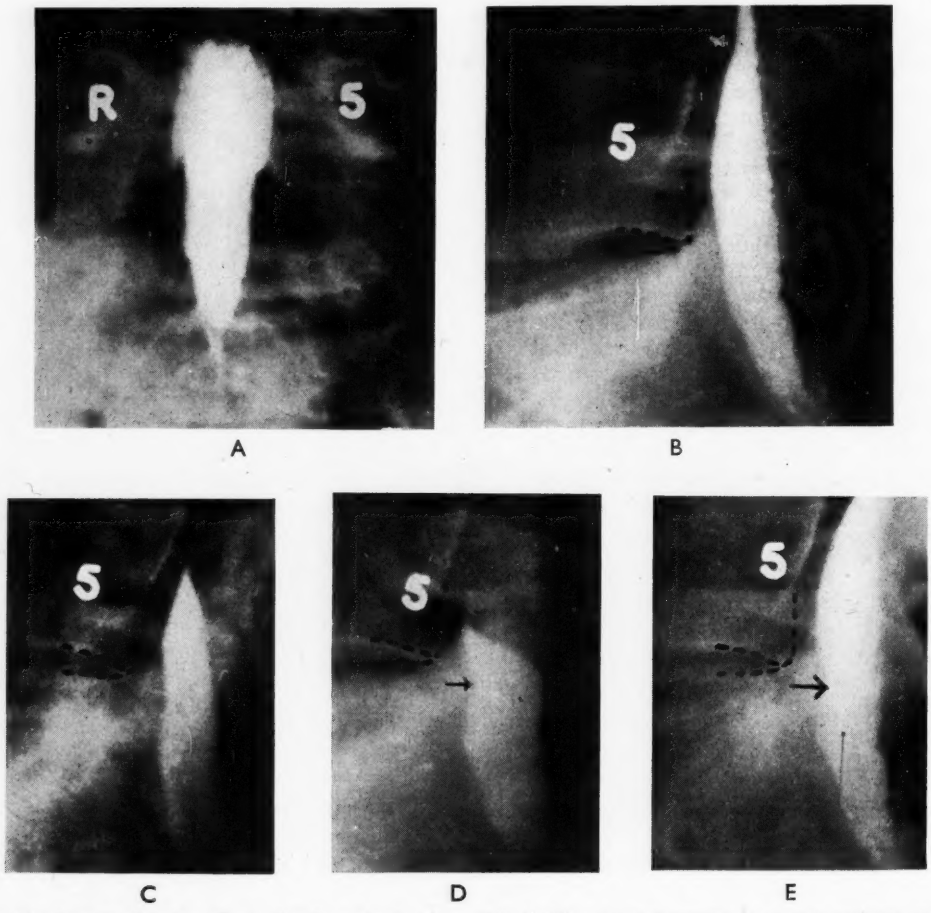


FIG. 210.—Myelograms showing effects of various stresses in a patient with a small intermittent central prolapse at lumbosacral space. A, B, Routine myelograms show no abnormality. C, Powerful manual compression applied to feet and shoulders with patient prone produces negligible change. D, When patient stands erect bearing 60-lb. weight on head, a significant defect appears. E, ' Hyperextension ' manœuvre with patient prone produces the most marked effect.
The anteroposterior view was not affected by any of these procedures. (× ⅔.)

the tilting fluoroscopic table compared with those present when he is on the operating table, and that these differences may account for some of the discrepancies which formerly seemed apparent between the myelographic and the operative findings. The patient on the operating table usually has all his voluntary muscular tensions lessened by anæsthesia, while if he is placed horizontally with his hips and lumbar spine flexed, the interlaminar spaces and posterior margins of the disk spaces are stretched. Under these circumstances the strains involving the intervertebral disk are at their lowest, and consequently the affected disk may not bulge when exposed surgically. However, the pathological disk may be made to protrude by means of a simple manœuvre, for if the operating table is broken so that the hips and lumbar spine are passively extended, the affected disk will usually bulge backwards (*Fig.* 208).

It is disk protrusions of this type which we describe as ' intermittent '. These are probably identical with the ' concealed ' disks referred to by Dandy (1941). Most cases of intermittent prolapse produce a positive defect on myelography, because the tensions resulting from muscular tone in the unanæsthetized patient are usually sufficient to make the disk protrude. Occasionally, however, myelography when performed in the ordinary way fails to reveal an abnormality, but if the patient who is lying prone then raises his head and shoulders off the table without disturbing the position of the lower half of his body, the prolapse becomes manifest. This manœuvre is comparable to the one we perform at operation, and its effects are well illustrated in *Figs.* 209, 210. In both instances the myelograms taken in the ordinary way appeared normal, whereas those taken after hyperextension outlined a central prolapse of appreciable size. In *Fig.* 209 it may be noted that the anteroposterior as well as the prone-lateral views show defects, whereas in *Fig.* 210 the defect was only seen in the prone-lateral projection. In *Fig.* 209 one may also note that not only did a midline disk prolapse reveal itself during the manœuvre, but also that an indentation appeared on the posterior aspect of the thecal column in the position of the ligamentum flavum. The considerable narrowing of the subarachnoid space between the two indentations, anterior and posterior, can be measured by the distance the opaque column has been forced upwards. The appearances also suggest how nerve-roots may be compressed between a disk prolapse in front and the ligamentum flavum behind.

Both Eaglesham (1944) and Childe (1945) have published myelograms indicating that weight-bearing, too, may accentuate the size of a disk prolapse, as judged by the observation that the myelographic deformity may increase when the patient stands erect compared with when he lies prone. Knutsson (1942) and Childe (1945) also have noticed that extension of the spine tends to accentuate myelographic deformities due to disk protrusions. We feel, however, that extension of the spine is the more potent factor, because in our experience it is easier to exaggerate a myelographic defect by this means than it is by weight-bearing (*Fig.* 210). Only a small proportion of prolapses are affected by either means, for often we have carried out our ' hyperextension ' manœuvre, and at other times have placed 70-lb. weights on a patient's head and shoulders, without appreciably affecting the size of the defects. We have even noticed that such procedures produced no effect on the prolapse of a patient examined with 24 hours of the onset of his first attack of pain.

It is too seldom realized that the strains imposed on the lumbar intervertebral disks vary greatly according to the posture and weight-bearing of the patient, and that the size of a disk protrusion may alter with the stresses affecting the disk.

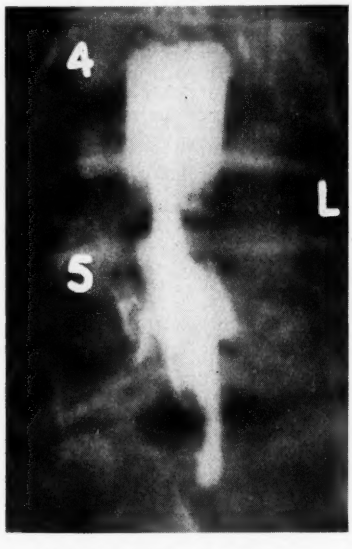


FIG. 211.—Myelogram showing disk prolapse at both 4th and 5th spaces. At 4th space central protrusion causing hour-glass constriction. At 5th space extrusion on right side displacing S.1 root pouch laterally and thecal column medially. (× ⅔.)

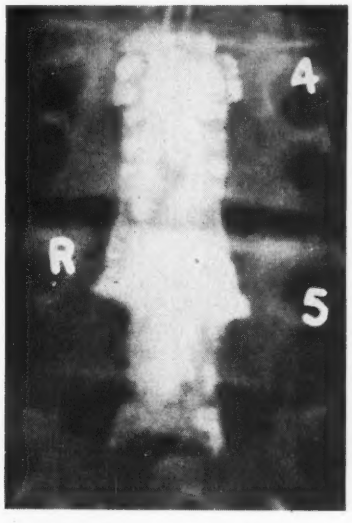


FIG. 212.—Myelogram showing orifice of L.5 root pouch on right side displaced slightly upwards, the result of an extrusion from the L.5 space. (× ⅔.)

3. Extrusions.—Extrusions may cause two characteristic myelographic appearances. They may displace root pouches instead of obliterating them, and they may cause defects in the thecal column at a

distance from the disk space. In *Fig.* 211, for instance, the first sacral root pouch is displaced outwards, while the second sacral root is deflected medially in its intrathecal course; at operation in this case a large extrusion of nuclear material was found wedged in the angle between the first sacral

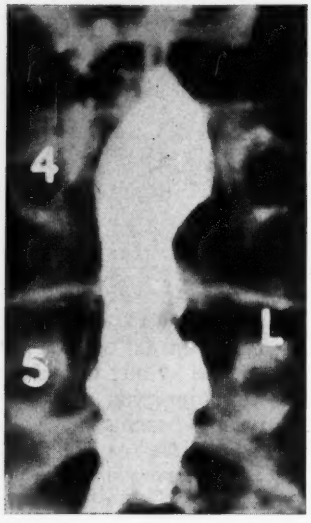


FIG. 213.—Myelogram showing lateral defect of thecal column on left side extending up to L.4 pedicle, caused by an extrusion from the 4th disk. (× ⅔.)

nerve-root and the theca. In *Fig.* 212 the orifice of the fifth lumbar pouch is elevated, and in this case an extrusion of the fifth lumbar disk had passed upwards alongside the theca to cause this deformity. In *Fig.* 213 there is a lateral defect of the theca at a level between two disk spaces; this was due to an

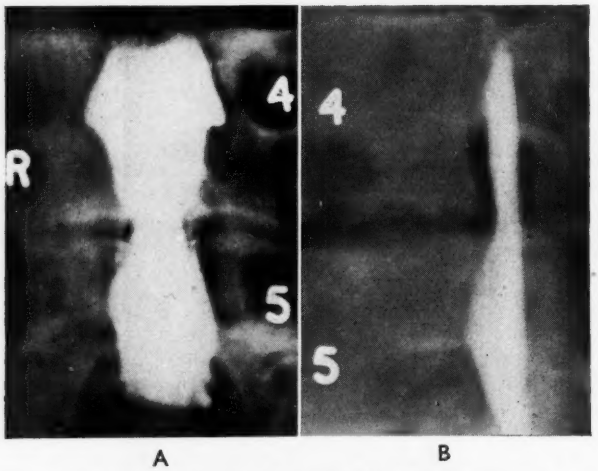


FIG. 215.—Myelogram of scarred disk causing symptoms. Note narrowed disk space. A, Anteroposterior view showing slight hour-glass constriction. B, Prone-lateral view showing that indentation of theca is caused by bony outgrowths. (× ⅔.)

extrusion of the fourth intervertebral disk which passed to become wedged between the theca medially and the fourth lumbar nerve and vertebral pedicle laterally. In *Fig.* 214 there is a wide-based defect extending from the fifth up to the fourth lumbar disk, associated with a displacement of the thecal column towards the opposite side; this was due to a large extrusion from the fifth disk.

4. Scarred Disks.—When a disk prolapse has been present for many years, secondary changes

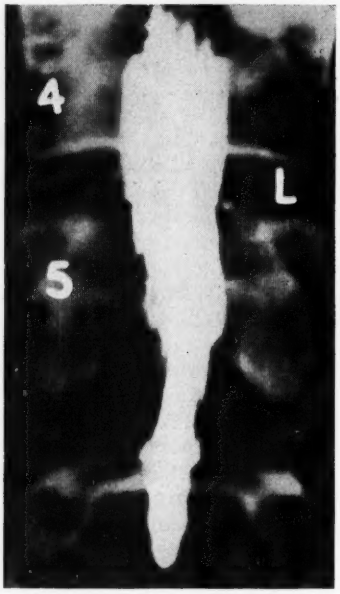


FIG. 214.—Myelogram showing wide-based defect extending from 5th up to 4th lumbar disk, caused by an extrusion from the 5th disk. (× ⅔.)

supervene. The disk space becomes narrowed, bony spurs develop at its margins, protruded nuclear material becomes absorbed, and the theca and the extrathecal nerve-root caught up by the scarring process becomes firmly adherent to the disk. Such a disk can usually be identified by scrutiny of the plain radiographs in conjunction with the myelograms. In the plain views the marked narrow-

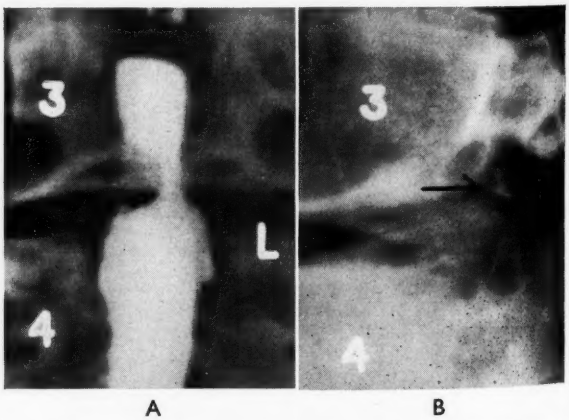


FIG. 216.—Scarred disk not apparently causing symptoms. A, Anteroposterior view of myelogram, showing large defect of thecal column. B, Plain lateral view, showing bony projection which caused this defect. (× ⅔.)

ing of the disk space, its sclerotic edges, and the osteophytic outgrowths are seen; while in the myelogram the prone-lateral views show that any indentation coincides exactly with the bony projections, and is not caused by a soft-tissue protrusion (*Figs.* 215, 216). Many scarred disks of this type do not cause symptoms.

C. INDIRECT EFFECTS OF DISK PROLAPSES

1. Swollen Intrathecal Nerve-roots.—Often the affected nerve-root is swollen for a variable distance proximal to the site of compression. This may be revealed not only by an absence of filling of the root pouch, but also by a band of radiolucence within the main thecal column which indicates the position of the swollen nerve-root (*Figs.* 217, 218). We have observed congestion and swelling of individual nerve-roots within the theca on a number of occasions, and each time have found it to be associated with compression of the nerve-root by a disk protrusion lower down. An analogous swelling of nerve-trunks proximal to the site of a compressing lesion has been observed experimentally by Denny-Brown and Brenner (1944).

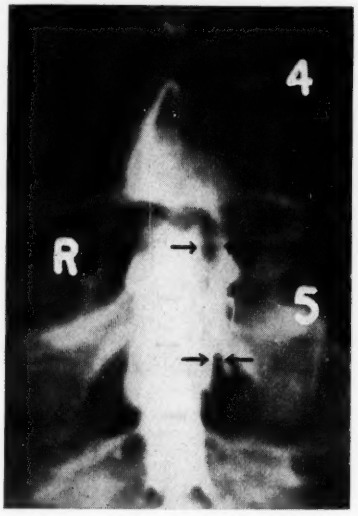


FIG. 217.—Myelogram showing multiple disk protrusions —4th space central, and 5th space left lateral. The outline of the swollen S.1 nerve-root can be traced within the thecal column for a distance of 3 cm. above the 5th space. At operation this nerve-root was twice the normal calibre. (× ⅔.)

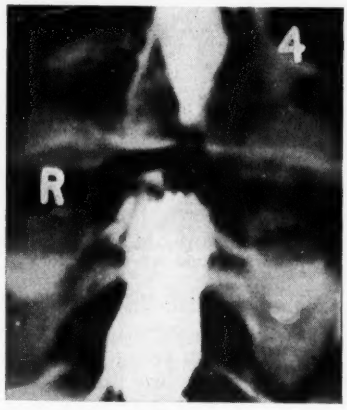


FIG. 219.—Block at 4th lumbar space due to leptomeningeal adhesions. Patient was in head-low position. Note serrated edge caudal to block. (A prone-lateral view showed serrations with no anterior indentation of theca at this level.) (× ⅔.)

2. Arachnoiditis.—Leptomeningeal adhesions matting together the elements of the cauda equina may develop occasionally at the level of a long-standing disk lesion, and produce an obstruction in the thecal column (*Fig.* 219). This 'block', however, presents certain features by which it can be distinguished from a block produced by a midline extrathecal lesion on the one hand, and a large intrathecal tumour on the other. Thus in lateral views there is no invagination of the anterior thecal wall such as occurs with midline disk prolapses, while in anteroposterior views the outline of the obstruction appears as a serrated edge, in contrast to the smooth edge which typifies a cauda equina tumour. This serrated edge is produced by seepage of contrast medium between the neighbouring roots of the cauda equina matted together with adhesions. A similar appearance is seen with blocks due to extrathecal compressions which crowd together the intrathecal nerve-roots.

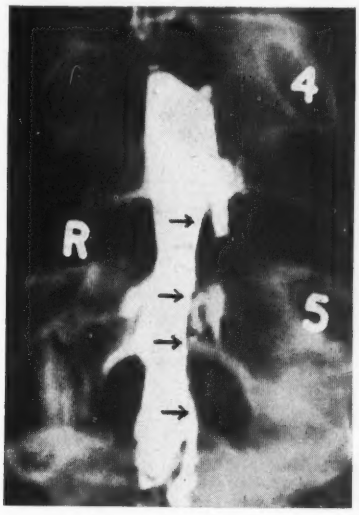


FIG. 218.—Myelogram showing effects of left lateral prolapse at 5th space. Here the large intrathecal defect was produced by the left S.1 nerve-root, swollen to four times its normal size. Its medial border is indicated by arrows. (× ⅔.)

D. MULTIPLE PROLAPSES

Multiple disk protrusions were demonstrated myelographically in 17 per cent of our cases (*Figs.* 211, 217, 220). In two other instances a second lesion was found at operation which had not been detected on myelography. In one of these, however, a complete block was present at the fourth space, preventing an examination of the lumbosacral space, where the second lesion was situated. This advance information of the number of lesions present has often proved useful at operation, and has guided the surgeon to disk prolapses which otherwise might have been overlooked. Any of the types of prolapse described may occur in combination. This is readily understood, since ordinary degenerative processes are the primary cause of the condition, and these processes affect to varying degrees all the intervertebral disks. Many, if not most, disk protrusions in the lumbar region do not cause pressure symptoms and therefore at operation only those protrusions should be dealt with which can be correlated with the clinical features of the case.

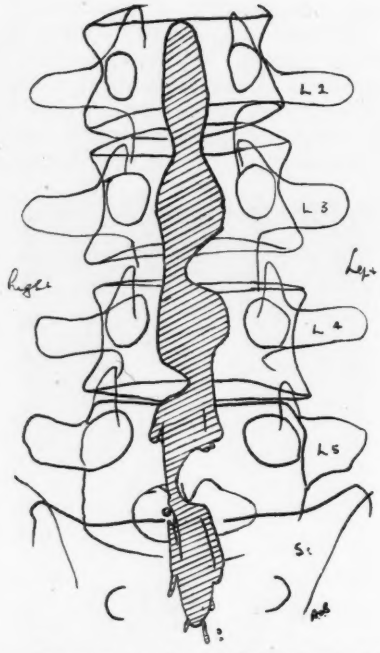


FIG. 220.—Composite tracing showing lateral disk protrusions at 3rd, 4th, and 5th spaces. (× ⅔.)

E. 'HYPERTROPHIED' LIGAMENTUM FLAVUM

Our experiences in regard to this condition has been largely of a negative character, but in view of the diversity of opinion which has been expressed concerning the nature and significance of these lesions, some observations made during myelography will be recorded. In this series we have found no myelographic evidence of hypertrophy of the ligamentum flavum in the lower lumbar region, nor did we establish its existence at operation. The myelographic appearances adduced by Towne and Reichert (1931), Hampton and Robinson (1936), and Brown (1938), as evidence of this condition were based upon anteroposterior views only, and show nothing which in our experience might not be caused by a disk lesion alone. Camp (1939; 1941) published prone-lateral as well as anteroposterior views of his myelogram, but the deformity which he believes to be characteristic of hypertrophy of the ligamentum flavum, is essentially similar to *Fig.* 209, C, D). Here a midline lesion of intermittent type was demonstrated at the lumbosacral space, although prone-lateral views revealed an indentation of the posterior aspect of the opaque column in addition to the anterior indentation due to the prolapse itself. This posterior defect, present only when the spine was extended, coincided with the position of the ligamentum flavum, which at operation seemed to be of normal size and texture. In our view this appearance is due partly to the bulging of normal ligaments when they relax on extension of the spine (Knutsson, 1942). A second factor, both in our case and in the one illustrated by Camp (1939; 1941), is that the disk prolapse has pushed the theca, here relatively mobile, backwards until it comes into contact with and outlines these ligaments.

These and other observations lead us to believe that true hypertrophy of the ligamentum flavum, if it occurs at all, must be a rare cause of symptoms. A prolapsed disk, however, may displace nerve-roots backwards to compress them against normal ligaments.

F. ARTEFACTS

Unless care is taken with the injection of contrast medium, curious and misleading appearances may occur which may render the myelogram worthless. These appearances are due to the presence of cerebrospinal fluid or of contrast medium outside the subarachnoid space, and may be caused either by faulty injection or by subsequent leakage. Leakage of cerebrospinal fluid through a puncture in the arachnoid gives rise to two types of artefact. If a large amount of fluid escapes into the subdural space, the subarachnoid space is compressed around the cauda equina and the opaque oil within it appears as a long thin column (*Fig.* 221). If only a small amount escapes, localized and irregular filling defects are produced in the thecal column (*Fig.* 222). Eaglesham (1944) has illustrated the mechanism of these subdural leaks of cerebrospinal fluid.

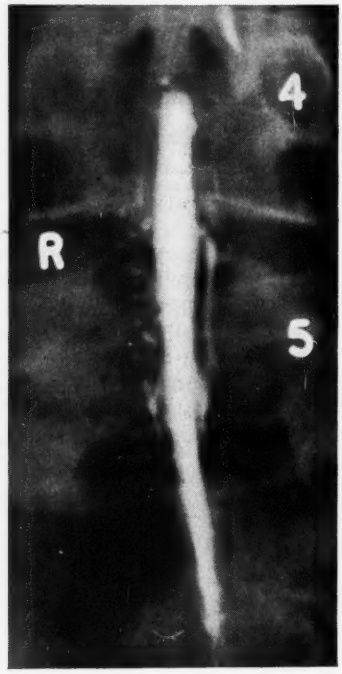


FIG. 221.—Attentuated thecal column due to generalized leakage of cerebrospinal fluid into subdural space. (× ⅔.)

If contrast medium leaks into the subdural space, it may remain in a mass, which on screening may be seen to move sluggishly along the spinal canal as the table is tilted, while any medium in the subarachnoid space rapidly overtakes and passes it (*Fig.* 223). If medium escapes into the extradural tissues it may pass along the track of the lumbar-puncture needle (*Fig.* 224). It may also seep along the epidural spaces, producing the startling appearance of *Fig.* 225.

Artefacts of minor degree may not vitiate the examination, but when they are gross myelography becomes impossible. In our series most artefacts occurred at a period when several persons employing varying technique were carrying out the injections.

As a consequence we learnt that several precautions must be observed :—

a. Lumbar puncture should be performed with the patient lying horizontal. If the patient is in the sitting position, the augmented pressure of the cerebrospinal fluid in the lumbar sac favours leakage into the subdural and extradural spaces.

b. The injection should not be made within several days of a previous spinal puncture. On occasions when myelography is repeated we have a gradual loss of medium from the subarachnoid into the subdural space, indicating that there has been a perforation in the arachnoid which has remained open for some days. As a result of these and other observations we believe that leakage of cerebrospinal fluid through puncture holes in the theca often occurs following lumbar puncture, and incidently is the fundamental cause of post-puncture headache.

c. When introducing the contrast medium it is important that a lumbar-puncture needle of fine calibre be used, and that the dura and arachnoid be pierced once only.

d. The examination should be made soon after the injection, the patient meanwhile remaining horizontal.

When these simple precautions were observed, we found that artefacts were eliminated, and have since performed myelography in more than a hundred cases without encountering them.

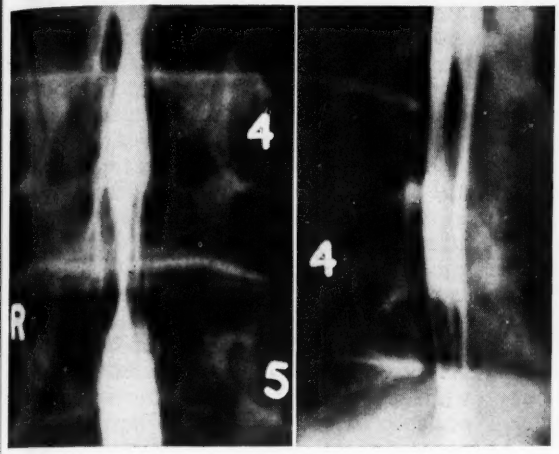


FIG. 222.—Effect of localized collection of cerebrospinal fluid in subdural space producing compression of subarachnoid space. The lateral view eliminated the presence of a disk lesion. At operation the nerve-roots appeared normal and there was no arachnoiditis. (× ⅔.)

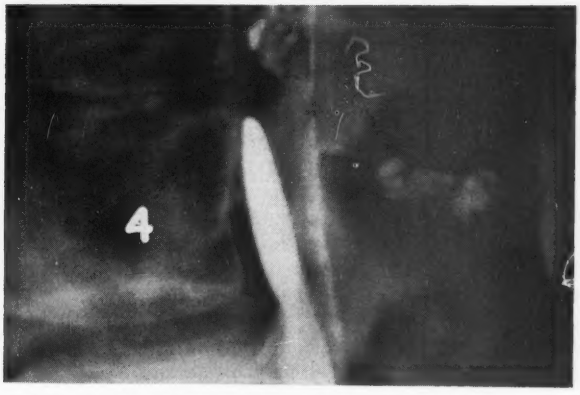
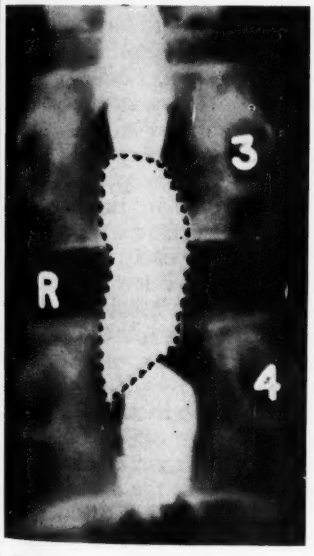


FIG. 224.—Escape of contrast medium into epidural space and along lumbar-puncture needle track. (× ⅔.)



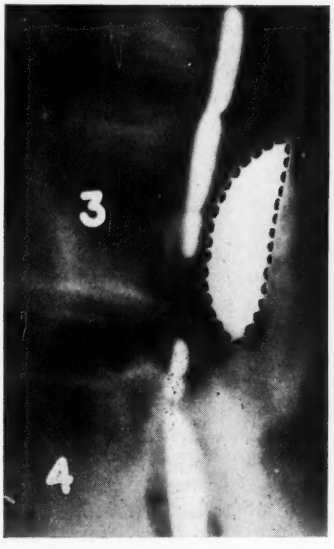


FIG. 223.—A localized collection of contrast medium in subdural space. The rest of the medium is in the subarachnoid space. (× ⅔.)

FIG. 225.—Permeation of contrast medium throughout epidural spaces. (× ⅔.)

DISCUSSION

Myelography can only become established as a routine procedure in the investigation of lumbar intervertebral disk lesions if it can be shown to be innocuous, to afford a reasonably accurate method of localizing lesions, and to give helpful information

which otherwise would not be available to the surgeon planning operation. We believe that myelography satisfies these requirements.

First Requirement: The Safety of the Procedure.—In none of our cases were any untoward effects seen which could be attributed to myelography. Strong objection has been raised to this procedure in that it involves the introduction into the subarachnoid space of a foreign substance, which may persist for a long time, if not indefinitely. Some authorities indeed have reported undesirable reactions, but we have not observed these. The reason may be that we employed only contrast media of established excellence (pantopaque and lipiodol Lafay), and if these were not available, we avoided the procedure rather than use substitutes of the harmlessness of which we were uncertain. Pantopaque we found to be slowly absorbed at a rate of two-thirds of its volume in a year. Lipiodol, however, is not absorbed. Until other and better substances become available, we feel reasonably safe in continuing to employ either pantopaque or lipiodol Lafay.

Second Requirement: The Accuracy of Procedure.—The second requirement concerns the accuracy of myelography in establishing the presence of a lesion. We feel that with experience the level and site of a prolapse can be deduced correctly; also the presence of multiple prolapses can be determined and sometimes the pathological type of the lesion forecast.

In order to study the value of myelography statistically we analysed our observations in the 86 cases out of our series which had technically satisfactory myelograms. Those cases were excluded in which the myelograms were worthless because of artefacts caused by a faulty technique or in which myelography was not performed because of temporary shortages of suitable contrast media. Of these 86 cases, 76 (88 per cent) showed myelographic appearances which were interpreted as pathological, while 10 (12 per cent) were considered normal. In all except 1 case one or more disk lesions were subsequently established at operation.

In the 76 cases in which the myelograms showed an abnormality, the anticipated lesion was subsequently found at operation in all but 2 instances. In the first of these two cases which proved exceptions, the myelogram had suggested that prolapses were present at each of the lower three lumbar spaces, one being of gross proportions and the other two only slight. At operation only the gross lesion was demonstrated, while the other two disks seemed normal. The operative exposure, however, was restricted to a peep-hole interlaminar approach at each space, and subsequently the patient, although improved, still had persistent though not troublesome backache. In the second case, one of intractable low back pain without sciatica, a central disk prolapse was indicated by myelography, but the disk when exposed through an interlaminar approach seemed normal. This was the only negative surgical exploration in our series, but as the patient was not benefited, it is possible here also that a disk lesion was overlooked at operation.

Thus when the myelogram is positive the presence and level of a disk protrusion are indicated with almost complete accuracy (74 out of 76 cases, or 98 per cent). In the 74 cases which were subsequently confirmed at operation, a prolapse was demonstrated myelographically at the fourth lumbar space in 46 cases and at the fifth space in 43 cases. In 14 multiple protrusions were disclosed, including 3 at the third lumbar space. Moreover, in nearly all these cases there was a high correlation between the site of the prolapse in relation to the theca and nerve-roots, as suggested by myelography, and that established at operation. The type of prolapse, however, could only be foretold approximately, but this information is rather a refinement of diagnosis, for the more fundamental information concerns level and site.

In each of the 10 cases in which the myelograms were negative a disk lesion was shown to be present at operation. Two cases of this group, however, were instances of multiple protrusions, in which only the larger lesion had been revealed radiologically (in one the major lesion caused a complete block which precluded examination of some of the lumbar spaces), while in a third case with an intermittent type of prolapse at the lumbosacral space a slight but definite degree of spondylolisthesis was present which may also have been responsible for symptoms. This reduces the number of negative myelograms to 7, and of these, 2 were examples of disk projection situated far laterally at the fifth lumbar space, while 5 were examples of intermittent prolapses. The 2 disk projections presumably did not affect the myelograms because they compressed the first sacral nerve-roots at points below the level of filling of the root pouches, and in this connexion the observation of Arbuckle, Shelden, and Pudenz (1945) that, if the thecal column is narrow, it may not be affected by a lateral protrusion, is also relevant. The 5 examples of intermittent prolapse (3 at the fifth space and 2 at the fourth) may possibly have been overlooked, because in none of them did we carry out the hyperextension manœuvre on the X-ray table, as we did not discover it until a later stage. Had we explored it earlier we might have had fewer normal myelograms. It must be remembered, of course, that most cases of intermittent prolapse produce filling defects on myelography similar to disk projections.

Thus in 12 per cent of cases in our series the myelograms were normal although disk lesions were present. It is possible that some lesions were missed through inexperience, but the fact remains that a negative myelogram does not exclude a disk lesion. On the other hand, a positive myelogram will demonstrate a disk lesion with great accuracy, not only as regards its level, but also as regards its site in relation to the theca and the nerve-roots.

Third Requirement: Useful Information.—The third question to consider is whether myelography affords useful information which cannot otherwise be obtained before operation. We have just shown how the procedure indicates with reasonable accuracy the level of the pathological disk, and so enables the surgeon to expose and explore it with the minimum of delay and trauma. In one of our cases, which had been explored elsewhere without benefit, the myelogram showed that the wrong disk had been exposed, and that the offending disk had not been disturbed. Preliminary myelography would probably have prevented this unfortunate

error. Similarly it discloses the presence of multiple prolapses, which in our series occurred in the lower lumbar region in 17 per cent.

A most useful piece of information is whether a disk prolapse is centrally or laterally placed. In our series approximately a third of the prolapses were centrally placed while two-thirds were laterally placed. We learnt from bitter experience that with a central prolapse it is often impossible to remove satisfactorily all the degenerated disk tissue from only one side of the theca, and that it is then necessary to expose and curette the affected disk from the opposite side as well. In the early stages of our work several of our patients after operation still had symptoms, such as severe backache or severe pain down the opposite leg. These were mostly examples of central prolapse where a unilateral procedure had been carried out. When at a second operation we came to explore the affected disk from the other side, we were often surprised to find a large amount of degenerated disk material still present in the disk space, and the removal of this usually relieved the symptoms. Consequently we now explore these central prolapses from both sides of the theca, and the knowledge which myelography affords of the situation of the lesion thus helps in planning operation.

Another advantage of myelography is that it assists in distinguishing tumours of the cauda equina which may be simulating disk lesions. We have met with only one example, an extrathecal hydatid cyst at the lumbosacral region which was causing sciatica. Love (1944), however, reports that at the Mayo Clinic, during a period when 100 consecutive patients underwent operation for protruded intervertebral disks, 8 cases of spinal tumour producing a 'disk syndrome' were encountered, and that a correct diagnosis was made pre-operatively in these cases largely because spinal puncture and contrast myelography were undertaken before operation.

In the absence of myelography a surgeon should expose both lower lumbar intervertebral disks from both sides of the theca if he is to avoid overlooking a lesion or dealing with one incompletely. We followed this practice when the myelograms were normal or were obscured by artefacts, but it is not a good policy to follow as a routine, because it means that frequently an unnecessary amount of surgical trauma is inflicted upon the vertebral column. To expose a disk adequately so that one can be certain it is normal, often requires the removal of the spine and lamina, for the exposure which is gained by an interlaminar approach is too restricted for the proper visualization of many central prolapses. Again, if a tumour is present the exposure has to be even wider. Knowledge beforehand of what he can reasonably expect to find at operation usually enables the surgeon to deal with a lesion with the minimum of trauma and delay.

CONCLUSIONS

For these many reasons we believe that myelography is worthwhile as a routine diagnostic procedure in lumbar intervertebral disc lesions. It is, however, important to realize that a myelographic defect indicating a disk lesion is not of itself sufficient justification for surgical intervention, for a disk protrusion may be symptomless. Thus we have examined the lumbosacral theca in cases in which myelography was being performed for the diagnosis of a lesion in the cervical and lumbar regions, and occasionally we have found evidence of a lumbar protrusion without associated symptoms. Again, in cases of sciatica which have resolved spontaneously, we have found that if myelography be repeated the defect indicating a disk lesion may persist long after all symptoms have subsided.

Myelography, therefore, should never displace clinical methods of investigation as the basis of diagnosis of intervertebral disk lesions. It serves, however, to confirm and amplify the clinical findings. We believe that myelography should play as important a role in the routine investigation of these lesions as does a barium meal in gastric disorders. We have been impressed by the fact that frequently myelography adds details to the pre-operative picture which subsequently are of assistance in treatment. Although media even more suitable than pantopaque will be introduced in the future, it seems unlikely that the principles underlying myelography will change.

We wish to thank Dr. William Sowerby, Chief of the Department of Diagnostic Radiology, and Mr. J. R. White, Chief of the Orthopædic Department at the Dunedin Hospital, for help and encouragement in this investigation, and those physicians and surgeons who have referred their patients to us. Also Miss V. Tregonning and the Staff of the Radiological Department for technical assistance.

We are indebted to the Medical Research Council of New Zealand for defraying the expenses of publication of this paper.

REFERENCES

ARBUCKLE, R. K., SHELDEN, C. H., and PUDENZ, R. H. (1945), *Radiology*, **45**, 356.
BRADFORD, F. K., and SPURLING, R. G., (1941), *The Intervertebral Disc*. Springfield, Illinois.
BROWN, H. A. (1938), *J. Bone Jt Surg.*, **20**, 325.
CAMP, J. D. (1939), *J. Amer. med. Ass.*, **113**, 2024.
— — (1941), *Med. Clin. N. Amer.*, **25**, 1067.
CHILDE, A. E. (1945), *Canad. med. Ass. J.*, **52**, 458.
COPLEMAN, B. (1944), *Amer. J. Roentgenol.*, **52**, 245.
DANDY, W. E. (1941), *J. Amer. med. Ass.*, **117**, 821.
— — (1943), *Ann. Surg.*, **118**, 639.
DENNY-BROWN, D., and BRENNER, C. (1944), *Arch. Neurol. Psychiat., Chicago*, **52**, 1.
EAGLESHAM, D. E. (1944), *Brit. J. Radiol.*, **37**, 343.
FALCONER, M. A. (1944), *N.Z. med. J.*, **43**, 58.
HAMPTON, A. O., and ROBINSON, J. M. (1936), *Amer. J. Roentgenol.*, **36**, 782.
KNUTSSON, F. (1942), *Acta radiol., Stockh.*, **23**, 431.
KUBIK, C. S., and HAMPTON, A. O. (1941), *N. Engl. med. J.*, **224**, 455.
LOVE, J. G. (1944), *J. Neurosurg.*, **1**, 275.
— — and CAMP, J. D. (1937), *J. Bone Jt Surg.*, **19**, 776.
MCKENZIE, K. G., and BOTTERELL, E. H. (1942), *Canad. med. Ass. J.*, **46**, 424.
MIXTER, W. J., and BARR, J. S. (1934), *N. Engl. med. J.*, **211**, 210.
SEMMES, R. E. (1939), *Yale J. Biol. Med.*, **11**, 433.
SKINNERS, B. M., and HAMBY, W. B. (1944), *J. Neurosurg.*, **1**, 117.
SOULE, A. B., GROSS, G. W., and IRVING, J. G. (1945), *Amer. J. Roentgenol.*, **53**, 319.
TOWNE, E. B., and REICHERT, F. L. (1931), *Ann. Surg.*, **94**, 327.

A CONCEPT OF PARALYTIC ILEUS: A CLINICAL STUDY

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"Oft the teeming earth is with a kind of colic pinch'd and vexed by the imprisonment of unruly wind within."—Shakespeare, Henry IV, Part 1.

I
INTRODUCTION

A gastro-intestinal complex—distension, gas pains, constipation—commonly follows many operations on the abdomen and sometimes operations in other parts of the body. As a rule, it is a temporary disturbance of intestinal motor function, but it may merge into a serious condition in which the patient is greatly distended, is vomiting continuously, and is completely constipated; that is, it may merge into a condition which is known as paralytic ileus.*

For the purposes of this study, 'paralytic ileus' is used to indicate all conditions characterized by distension, constipation, vomiting, and lack of bowel movements.

The present article is a study of this gastro-intestinal complex undertaken to see whether it might be prevented by any change in routine pre- and post-operative treatment, and it is also a study of the treatment of developed paralytic ileus.

From this literary, clinical, and experimental study it is hoped to establish these main points:—

1. That 'true' or idiopathic paralytic ileus is rare;
2. That many cases which are regarded as 'true' or idiopathic post-operative paralytic ileus are caused by intra-abdominal infection of some sort;
3. That there are three definite stages of ileus:—an early, a middle, and a late—paralytic—ileus;
4. That intestinal movements are present in the early stages of the ileus, but in the late stage they are absent;
5. That a regimen of treatment based on each stage should be laid down.

MATERIAL STUDIED

The study was approached from several different angles after available recent literature had been studied.

1. The records and histories over a period of six years of 43 consecutive intestinal intubations with the Miller-Abbott tube, and a number of cases seen in consultation, but which had not required intubation, were examined and analysed.

2. Observations on intestinal movements, and the return of intestinal movements after ileus, with recording apparatus connected to the balloon of the Miller-Abbott tube were made, with and without the use of drugs. At first a recording tambour was used to record the movements on smoked paper fastened around a pie-dish mounted on an electric clock which revolved once in twelve hours. However, as most of the patients were desperately ill and required considerable nursing attention, many of the records were brushed off by the nursing staff. Then a commercial clock-face ink-recording barometric type of pressure recorder was used, and, with the pointer loaded, was found to be satisfactory and "nurse proof". Most of the records included are from this apparatus, in an effort to demonstrate instrumentally what was observed clinically. Difficulties of recording in these sick patients were added to by the fact that coughing, vomiting, straining, etc., and all attempted movements caused sudden changes of intra-abdominal pressure which were recorded as "spikes" on the slowly-moving drum, and in *Fig.* 229, *a*, the effects of these factors on the records is shown.

3. In two patients who had ileostomies, records were made from balloons placed in the ileostomies.

4. X-ray studies were made of the effect of some drugs on bowel motility in normal subjects.

5. Auscultation of the abdomen was made on all cases seen, and also on a number of cases in the immediate post-operative phase following appendicectomy and other abdominal operations, the return of movements being particularly noted.

6. Observations were made on a number of patients in whom gross soiling of the peritoneal cavity had caused paralytic ileus, and in whom morphine was given prophylactically.

7. The effect on bowel movement of heat on the abdomen and turpentine stupes, and of some of the commonly used drugs, was also studied.

8. In two patients with ileostomies and a separate "defunctioned" colon, the effect of colonic irrigation on small bowel movement was observed.

9. In two patients in whom the Miller-Abbott tube had been introduced through a jejunostomy, the effect of gastric distension on small-bowel motility was observed.

CAUSES OF ILEUS—AND OF PARALYTIC ILEUS

A table of the causes of ileus has been put forward by Ochsner and Gage (*see* Appendix). The classification of the causes of paralytic ileus, however, is perhaps more appropriately set out under the three following headings: (1) 'True' or idiopathic paralytic ileus; (2) Paralytic ileus arising from medical diseases; (3) Paralytic ileus the result of surgical operations.

1. 'TRUE' OR IDIOPATHIC PARALYTIC ILEUS

Idiopathic or paralytic ileus of unknown cause may be due to over-action of the sympathetic nervous system, and thus it may arise (*a*) as a result of stimulation, or (*b*) following reflex inhibition.

* Ileus is defined vaguely as 'a kind of colic', and 'iliac passion' as intestinal colic. Thus the word has wandered far from its original or earlier meaning, for a 'paralytic colic' seems a contradiction.

a. As a Result of Stimulation.—This is probably the cause of the frequent development of paralytic ileus in cases of retroperitoneal hæmatomata (Karabin, 1942). This origin is exemplified in the following case :—

Example 1.—A male, aged about 30 years, was hit by a car and suffered a fracture of the pelvis. When admitted he was very shocked and was given intravenous serum. Catheterization showed that there was blood in his urethra, but the catheter passed into his bladder. On the third day he became greatly distended and there was no result from a rectal tube, pituitrin, or enemas. He vomited copious quantities of brownish fluid. A Miller-Abbott tube was passed and from the various tests (John Devine, 1945) it appeared to be beyond the pylorus. However, he became completely irrational and pulled it out. The tube was put back several times in the night, but the next morning he died. Post-mortem examination showed a retroperitoneal hæmatoma and enormous distension of the small and large bowel with clear intraperitoneal fluid. There were no evidences of peritonitis.

b. Idiopathic Paralytic Ileus caused as a Result of Reflex Inhibition of Gut Movement.—This probably occurs through the sympathetic system. The reflex may arise from a slight blow on the abdomen, peritoneal irritation, an abscess, acute appendicitis, or even from just the opening of the abdomen.

In this type of paralytic ileus it has been the experience of the author that a spinal anæsthetic initiates peristalsis—at any rate temporarily—and that it is not inhibited during the subsequent operation.

In addition to the types above described, there is, also, following all abdominal operations, a reflex inhibition of intestinal movements. Abdominal auscultations carried out by the author, after operations of a routine type, showed that the first sound to be heard in the abdomen occurred between three and eight hours following the operation ; in other words, there was a temporary phase of ileus. Moir (1943) points out that opening of the abdomen itself produces a period of an " active " phase of ileus. This has been confirmed by others. Davis and Hansen (1945) gave barium to patients before operations for appendicectomy performed under spinal anæsthesia. They showed that following the operation there was a definite decrease in intestinal movements and a delay in emptying the stomach. Wakim and Mann (1943) found that ether anæsthesia and exploratory laparotomy stopped alimentary tract movement for at least four hours. Ether anæsthesia and an operation for bowel anastomosis caused cessation of intestinal movement (i.e., ileus) for at least twenty-four hours ; it was seventy-two hours before weak peristaltic waves were recorded ; but after the fifth day no detectable differences could be seen between records of intestinal activities made prior to operation. Even then, however, there was still some inhibition of intestinal movement in the sectioned loop.

The time of recovery from this post-operative period of active ileus varies greatly, depending on the operative procedure, the anæsthetic among other factors. Observations by the author suggest that recovery from this ' active ' ileus closely corresponds to the recovery from ' paralytic ' ileus, and for this reason deserves careful study.

It is probable that a continuation of this very variable period of ' active ' ileus, enhanced by either a mild unnoticed post-operative infection, or non-physiological treatment, may, in a number of cases, be the origin of complete post-operative paralytic ileus.

A post-operative ileus is frequently found associated with a post-operative difficulty in voiding urine. Observations of the author show that urinary retention and post-operative ileus tend to occur in the same type of patient—i.e., one who may have a hyperactive ' sympathetic ' system. The following case illustrates such an association :—

Example 2.—Seven days after an operation for a left inguinal hernia, a man aged 45, who had had to be repeatedly catheterized, became distended. He began to vomit fæculent material and had hiccup and a dry brown tongue. A Miller-Abbott tube was passed, and in the first twenty-four hours 38 oz. of fæculent fluid and a great deal of gas were withdrawn, relieving his distension. Even then he still had some difficulty with micturition, but his convalescence was uneventful.

In this case when the tube was 3 ft. past the pylorus peristalsis was very slight, but following one pint of serum intravenously peristalsis became much more marked and frequent and the tube drained much greater quantities of material and air.

A full bladder or inflammation or stimulation due to other causes of the kidneys or bladder may cause inhibition of movement of the ileum and increase of gas in the intestine. (Allen and Jamison, 1940 ; Sviér and Mann, 1943.)

The following case illustrates the immediate cessation of bowel movements resulting from placing gauze packs in the abdomen.

Example 3.—A woman of 25 years had a Gilliam suspension of the uterus performed. Following operation, intraperitoneal bleeding occurred, and two days later the abdomen was reopened but no bleeding point was found. The pelvis was packed with gauze and the patient transfused. When seen some six hours later she had become markedly distended. A tube was substituted for the packing and the distension became noticeably less. As auscultation revealed no borborygmi a Miller-Abbott tube was passed. She was much improved for a week. Then she developed fever, rigidity, slight jaundice, and again distension. Infection of the intraperitoneal blood was assumed, and again the Miller-Abbott tube was passed and chemotherapy commenced. She recovered from this phase, but on the fourteenth day began to get definite intestinal colic, and a mechanical obstruction of the small bowel was correctly diagnosed. Before the operation a Miller-Abbott tube was introduced through a jejunostomy, and when the distension had thus been relieved the abdomen was opened and multiple dense adhesions were encountered. Division of these and ileostomy were performed. She died two days later from pneumonia.

Cathartics have an effect on the movements of the small bowel. Alvarez (1918) has shown that following purgation there is a period of intestinal atony and immobility. If, in pre-operative preparation, this period is allowed to coincide with an intestinal operation, the depression of bowel motility and therefore the post-operative ileus is increased. It is possible that fewer cases of post-operative

paralytic ileus occur now because routine purgative pre-operative preparation is not carried out.

A paralytic ileus, possibly of idiopathic type, may occasionally follow the most trivial injuries or procedures, and it is impossible to say whether it is nervous in origin. For example :—

Example 4.—A middle-aged woman with a long history of hysteria and neurosis had a minor orthopædic procedure performed on her foot. Ten days later she gradually developed abdominal distension and occasionally vomited. The abdomen became silent. Pituitrin and enemas failed to relieve the distension, so a Miller-Abbott tube was passed and gas and fluid evacuated. It was removed after several days, and though she sometimes had a return of the distension, all investigations (including barium meal and enema) were negative. She has remained reasonably well for the last six months.

The author has seen paralytic ileus follow the application of a plaster hip spica for a compound fracture of the femur due to a bullet wound.

It is usual in all cases of paralytic ileus of the 'idiopathic' or 'peritonitic' type that the distension is relieved by the Miller-Abbott tube and recommencement of bowel movements carry down the tube. This removal of distension results in the return of local bowel movement. This is agreed by all observers and has been verified personally. A logical inference from this is that in some cases the distension may be at least the initial cause of the lack of motility and not, as is usually stated, that bowel paralysis is secondary to the distension, though, of course, the balloon on the tube is a powerful stimulus to bowel contraction. This fundamental conception does not explain cases of post-operative 'active' ileus where there may be no distension and no movement. It does, however, seem probable by analogy, as overstretching of other involuntary muscular mechanisms such as in the bladder and sphincter ani is followed by a temporary or partial paralysis of bladder or sphincter. Another example is the improvement in the heart's action if it is dilated by venesection.

If this reasoning is true, then the key to the prevention of post-operative ileus is the prevention of post-operative distension.

Paralytic ileus may occasionally be due to hypoproteinæmia, as when the serum protein concentration is low the motility of the bowel is also considerably lowered, possibly due to bowel œdema. After serum transfusion the motility increases. (Leigh, 1942; Barden, Thompson, Ravdin, and Frank, 1938; and Devine, 1938.)

2. PARALYTIC ILEUS CAUSED BY MEDICAL DISEASES

A paralytic ileus can be brought about by any form of general infection or general toxæmia. It also occurs in uræmia, the late stages of pneumonia, and in the terminal stages of other medical diseases, e.g., it may be the terminal stage of a case of liver failure, as in the following case :—

Example 5.—An elderly man had an operation for the removal of stones from the common bile-duct. He had been continually jaundiced for one and a half years and intermittently for fifteen years. After the operation he developed liver failure, became distended, and had no bowel movements. Distension was removed by the passage of a Miller-Abbott tube, but he died in delirium a week later.

3. PARALYTIC ILEUS CAUSED BY SURGICAL DISEASES

It may be the result of :—

1. Intestinal obstruction, mechanical or otherwise, because long-continued distension leads to bowel paralysis.

2. Paralysis of the bowel from the presence in the peritoneal cavity of pus, urine, blood, bile, gastric, duodenal, or intestinal contents.

In some cases in the series studied, one loop of the intestine was bathed by pus in the pelvis and produced intestinal obstruction from the local paralysed segment of bowel which, after some time, caused paralysis as a result of ascending distension of the remaining intestine. This was found to be a more common type of 'paralytic ileus' than the so-called 'idiopathic' type, and complete recovery followed drainage of the usual cause, viz., a pelvic abscess. In the early stages of these cases borborygmi were increased above, but in the late stages the abdomen was silent. A paralysed loop of intestine was causing obstruction.

The following case histories illustrate conditions of ileus which were apparently infective in origin. One also illustrates the fact that the establishment of a condition of ileus followed stimulative treatment :—

Example 6.—A middle-aged married woman had had a hysterectomy five days before I saw her. At the operation it had been found that she had an infected polypus in the cervix and this was closed over with difficulty. She had a slight rise in temperature after her operation and began to get distended on the third day. She was given pituitrin with only a small result, but with relief of the distension. She again became distended, and again was given pituitrin with the same result. Each time she became a little more distended after the pituitrin. By the fifth day she was very distended, and had a resonant abdomen. She had great difficulty in breathing and looked very ill. She had been having intravenous fluid, but no serum.

On examination she still had many borborygmi, and it was concluded from the history and physical signs that she probably had some loops of small bowel lying in an infected area in the pelvis. A Miller-Abbott tube was passed and 240 oz. of fluid was withdrawn in twenty-four hours. Distension was thus relieved and serum was given at the same time; her general condition improved. The following day a posterior colpotomy allowed pus to be drained from the pouch of Douglas. Following this, her convalescence was uneventful except for pneumonia, which was treated with penicillin.

While the tube was in position, a recording apparatus was connected to the balloon lumen and it was found that penicillin had no effect on bowel movement, but barbiturates considerably lessened them, and no effect could be observed with $\frac{1}{6}$ gr. of morphine.

Example 7.—A woman, 30 years of age, had had a miscarriage many years before, and following this had been unable to have children. A salpingostomy was done, much against the wish of the gynæcologist who performed the operation, but the patient insisted on taking the risk of opening up the old infected tissue. Following the operation she had fever varying between 99·4° and 100·2°, and during the first two days vomited gastric contents. On the third day she began to vomit intestinal contents.

When I saw her on the fifth day she was vomiting dark-brown fæculent material in large quantities. She was distended and on auscultation her abdomen was silent. She looked very ill. Per vaginam she had tenderness over the uterus, but no mass was palpable. The condition was diagnosed as one of infection of a loop of bowel in the pelvis which had caused complete paralytic ileus. A Miller-Abbott tube was passed with relief of the distension, and at the same time sulphonamides and penicillin were commenced. After forty-eight hours the balloon of the tube was deflated and the tube withdrawn to the 2-ft. mark. She again became distended and again the balloon was inflated and allowed to pass through the intestine. Five days after the first intubation, that is, ten days after operation, she commenced to have frequent loose bowel action. Convalescence from then on was uneventful.

Example 8.—A middle-aged woman with a placenta prævia had had a lower segment Cæsarean section done six days before being seen. After this she became distended and streptococci were cultured from the vaginal discharge. She had a high temperature from the third to the sixth day and penicillin had made no difference to her condition. When first seen she was very distended, slightly jaundiced, with a pulse of 130, and was hiccuping and apathetic in appearance. There were no sounds to be heard at all in the abdominal cavity. The serum protein was 5·7. She was given three litres of serum and a Miller-Abbott tube was passed. This drew off 180 oz. of fluid in the first sixteen hours. Her condition improved rapidly and the convalescence was uneventful.

Example 9.—A woman about 55 years of age had had an operation for bilateral malignant ovarian cysts five days before being seen. Following the operation she had a steep rise in temperature and on the third day commenced to have distension and vomiting. By the seventh day she was very distended, had difficulty in breathing, and was slightly cyanotic in colour. The serum protein was 6·4. On auscultation of the abdomen many gurgling sounds were heard high up, but the hypogastrium was silent. It was concluded that she had a pelvic peritonitis with some loops of small bowel in it, and she was given two litres of serum and a Miller-Abbott tube was passed. Penicillin and sulphonamides were also given. In the first twelve hours 40 oz. of fluid and a large amount of gas were aspirated by the tube and her distension disappeared by next morning; her bowels commenced to act the next day. Convalescence was uneventful.

Here is an example of ileus due to general peritonitis :—

Example 10.—An elderly man who had had an excision of a diverticulosis with five sinuses, had an obvious peritonitis for which chemotherapy and intubation were employed, but despite the fact that the distension was relieved and sounds were again heard in a previously silent abdomen, death occurred on the tenth post-operative day.

The following case also illustrates ileus from peritonitis, and the fact that wash-outs through an ileostomy, while increasing bowel movements, may actually increase distension when given at the wrong 'stage' of bowel movements.

Example 11.—A middle-aged man who had had a carcinoma of the ascending colon developed a leak from the suture line of the ileum to the transverse colon. The abdomen was opened and the leaking area exteriorized and ileostomy done. A Miller-Abbott tube was then passed to relieve his distension, and, though it was successful in this, he developed auricular fibrillation the next day, became irrational, and finally died. It was noticed in this case that wash-outs through the ileostomy markedly increased his distension.

THE PATHOLOGICAL PHYSIOLOGY AND THE PATHOLOGY OF PARALYTIC ILEUS

Some idea of the frequency of paralytic ileus as a cause of death may be gathered from the fact that from the Royal Adelaide Hospital 10 cases were found in 1000 post-mortem examinations. (1944, *Med. sci. Arch. Adelaide Hosp.*)

At post-mortem examination all that is found in a case of 'true' paralytic ileus is some thin intraperitoneal fluid and a large mass of distended small bowel filled mainly with gas, and the length of the intestines is markedly diminished.

In paralytic ileus the bowel wall is thin, dusky, and œdematous, suggesting obstruction to the venous return. Distension by itself can produce venous obstruction. A pressure above 55–65 mm. Hg within the gut causes pressure on the veins which encircle the ileum, interferes with the absorption of gas and fluid, and leads to more distension

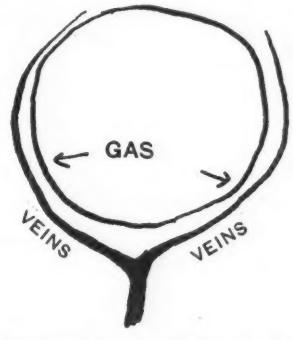


FIG. 226.—To show how increasing distension causes pressure on the veins and interferes with absorption from the bowel.

(Wangensteen, 1942). A vicious circle is thus brought about (*Fig.* 226). Greater distension can even cause arterial compression.

The gas in the bowel of paralytic ileus is partly nitrogen, coming from swallowed air (Wangensteen, 1942), and partly carbon dioxide which is normally 4 per cent. The percentage of nitrogen is from 60 to 80. An important relevant observation is that the normal small intestine can absorb about 2500 c.c. of carbon dioxide and about 1300 c.c. of other gases per hour (McIver, Redfield, and Benedict, 1926; Davis and Hansen, 1945). These workers showed that most of the gas in the bowel following an anæsthetic is swallowed air. They proved that barium given to patients before operations for appendicectomy was not passed out of the stomach as quickly as when no operation had been performed. Thus they come to the conclusion that after operations gas is not passed along the bowel for expulsion, and that it accumulates and gives rise to pain.

The fluid in the bowel of paralytic ileus is thin, brownish, and flaky. The greater part of it is probably poured into the intestinal tracts in the form of saliva, gastric juice, bile, and pancreatic and intestinal secretions. This normally amounts to

from 7½ to 10 litres per day (Rowntree, 1922; and Gamble, quoted by Bockus, 1944.) This fluid is toxic. Injection of either the contents of the normal or of the obstructed bowel into the veins of experimental animals can be fatal. This, however, does not prove that a toxin is the cause of death in cases of paralytic ileus.

THE EFFECTS OF DISTENSION OF THE BOWEL

The effects of distension can be local or general.

Local Effects.—Multiplication of gas-forming bacilli, such as *B. Welchii*, occurs (Williams, 1927). One local result of distension is that the capillaries of the intestinal mucosa become more permeable and some of their contents transude into the peritoneal cavity. Here the water is absorbed by the peritoneum and a fluid, which becomes more and more like plasma in its protein content, is the result. Thus loss of plasma from the circulation may be considerable—and loss of plasma decreases bowel motility, probably due to œdema of the bowel. Thus a vicious circle is formed. (Barden, Thompson, Ravdin, and Frank, 1938; O. C. Leigh, 1942.)

Fluid is not only lost from the intestine, but since in most cases of paralytic ileus the pylorus also is inhibited or paralysed, vomiting of intestinal contents

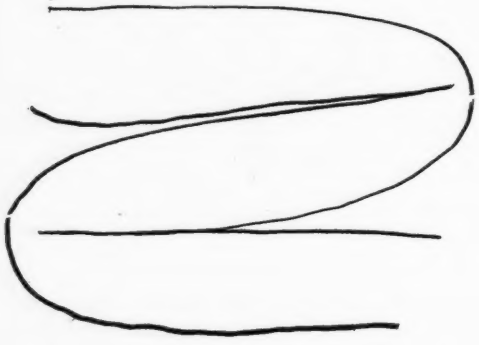


FIG. 227.—To show how obstruction occurs at each end of loops of small bowel in marked distension.

occurs. For instance, in *Case* 1, in which multiple ileal obstructions were present, fluid which could be recognized was introduced through the suction lumen of a Miller-Abbott tube, which had been radiologically proved to be in the mid-ileum, and was later vomited by the patient.

The vomiting and loss of intestinal contents in turn bring about loss of sodium chloride. Thus is started a deleterious vicious circle: the loss of large amounts of the irreplaceable sodium ion causes a shrinkage in the blood-plasma volume; this loss leads to a diminution in the volume of intercellular fluid; and even if water is given by mouth, salts will be secreted into it, to be again lost in the vomitus. The blood becomes hypotonic; the kidneys automatically correct this by excreting water and thus add to the dehydration. Therefore, the hæmoconcentration and dehydration caused, bring about a circulatory failure which primarily is attributable to the loss of sodium (Bockus, 1944).

One local effect of distension is that of itself it can cause further obstruction by the kinking of the distended small bowel at the apex of the loops (Ashcroft and Samuel, 1940; Ochsner, 1937). (*Fig.* 227.)

A further effect of distension is that the longitudinal and circular muscles are greatly weakened in their action by distension. This is mechanical, and the pressure developed by the muscles decreases in proportion to the increase of the radius. (R. D. Wright, 1946.)

General Effects.—There is a general effect from local distension. Local distension of a loop of small bowel causes vomiting, anorexia, and inhibition of movement in the remainder (Youmans, 1944; Burget, Martzloff, Suckow, and Thornton, 1930). If the distended loop of bowel has been denervated, the distension causes no vomiting and no inhibition (Antoncic and Lawson, 1941). In dogs, distension of an isolated loop of small bowel caused death in eight days, but the dogs could be kept alive longer by giving saline infusions.

Patients with paralytic ileus have a great thirst because of their dehydration and loss of salt, and therefore much of the fluid found in the bowel has been taken by mouth.

There is also a difference in the flow of bile. The author has noticed that in most of his cases of paralytic ileus there was little or no sign of bile in the aspirated fluid. This may have been due to extreme dilution of bile, or to the fact that distension of the intestine decreases the bile flow (Schnedorf and Orr, 1941). Here again is a vicious cycle, for bile administered either through the duodenum or injected into an isolated loop of intestine greatly increases its motility (Haney, Roley, and Cole, 1939).

It is characteristic of a fully developed paralytic ileus that the large intestine is also involved. This has been shown by X-ray studies (Hunt, 1944), and also it was the experience of the author that in cases of fully developed paralytic ileus enemas were retained or returned without result. This is illustrated in the following case:—

Example 12.—An interval appendicectomy was performed. Thirty-six hours after the operation the patient began to suffer from severe colicky, central-abdominal pains, which were unrelieved by ¼ gr. morphine. The colic occurred once every one to three minutes and lasted half a minute. Severe distension quickly developed, and there was some difficulty with micturition. At this stage (the 'second stage', referred to later) two enemas were given, the first glycerin and soapy water and the second milk and treacle. Both enemas were of one to one-and-a-half pints, and both were retained. Neither gave relief from the colic. Next day the colic had ceased, but purgatives, including two doses of calomel, were given without results. On the eighth day the bowels were opened with an enema and from that time on distension and discomfort were relieved. This case is quoted to show that in incipient paralytic ileus the colon is involved and that enemas do not act or remove distension or relieve colicky pains.

The pathology of paralytic ileus, arising as a result of infective or mechanical causes, will be associated with those pathological changes characteristic of inflammatory abdominal foci and of mechanical obstruction.

THE VARIOUS TREATMENTS OF PARALYTIC ILEUS

The methods of treatment recommended both for prophylaxis and in the developed disease have been many, varied, and confusing.

The ' whippers-up ' or ' stimulators ' of the bowel form one school. Their treatment is based on the fact that in this condition the bowels do not open, and, therefore, if the bowels are opened the condition will be cured. However, the absolute constipation (lack of defæcation and passage of flatus) is only a symptom and not the disease. Stimulation of the bowel was formerly the method of treatment, and is only now going into disrepute as a general measure. Thus, Stout (1943) advised early parasympathetic stimulants as prophylactic treatment against paralytic ileus, e.g., 0·5 mg. prostigmine, or 1 mg. thiamine hydrochloride every four hours for ten doses as a prophylactic. Marden and Williamson (1939) recommended prostigmine methylsulphate, 1 c.c. of 1–4000 solution, as a prophylactic, and 1 c.c. of 1–2000 solution to combat fully developed paralytic ileus.

Ochsner, Gage, and Cutting (1930) concluded that physostigmine was the only drug which had a definite place in the treatment of intestinal atony. Hartman and Dock (1927), on the other hand, supported the use of choline.

Treatment by stimulation with cathartics increases the movement of the normal small bowel (Oppenheimer and Mann, 1941), but many personally observed cases show that during an established ileus they have no effect whatsoever.

The following cases illustrate the ineffectiveness of cathartics in paralytic ileus (if cathartics ' work ', the patient has not got paralytic ileus) :—

Example 13.—A middle-aged woman had had a hysterectomy performed. On the second day she was given an enema without result; then cascara and paraffin and magnesium sulphate, without either results or abdominal colic. Next day more cascara and another enema were given, again without result. When seen on the fourth day she was very distended, and on auscultation of her abdomen occasional sounds were heard. She recovered after $\frac{1}{6}$ gr. morphine had been given four-hourly for three days. Her bowels were then opened by an enema.

(*Example* 17 also illustrates the ineffectiveness of cathartics in ileus.)

Among the stimulant forms of treatment must be mentioned the giving of intravenous hypertonic saline solution as advocated by Ochsner, Gage, and Cutting (1933), as this had been shown to increase peristalsis (Hughson and Scarff, 1924 ; Kleitsch and Puestow, 1939).

Pitressin and pituitary extracts have been extensively given for ileus.

Yet another school favour the treatment of ileus by " resting of the bowel " ; $\frac{1}{6}$ gr. morphine given every four hours is said to " rest " the bowels. (*See* p. 167.)

Another treatment of ileus has been the use of spinal and splanchnic anæsthesia, which has been shown at least temporarily to increase peristalsis. (David and Loring, 1930 ; Wagner, 1919 ; Novikov, 1939.)

Ileostomy is not a physiological method of treatment of paralytic ileus, as it drains only the segments in which it is placed ; yet a number of reports of the successful use of ileostomy are reported in the literature. But the bowel is not acting and therefore will not drive the contents down to the ileostomy opening ; and also in a paralytic ileus the folding of the distended small bowel gives acute obstruction at the apex of each fold. (*See Fig.* 227.)

The difference between paralytic ileus and obstruction is clearly shown in the radiograph of the two conditions. In paralytic ileus small pockets of gas are seen scattered all over the abdomen and the colon is also distended ; whereas in acute obstruction continuous loops of distended bowel are seen. Thus, when paralytic ileus is cured the ileostomy acts furiously and well ; but it is not the ileostomy which has cured the conditions.

Example 14.—Paralytic ileus followed the making of an ileostomy as a preliminary to colectomy. In this fully developed condition the patient would not tolerate the Miller-Abbott tube in the pharynx, so it was introduced through a jejunostomy under local anæsthesia. No peritonitis was found when this was being done. The abdomen was finally decompressed, but the patient died on the tenth post-operative day from a pulmonary embolus.

In several cases the ileostomy did not work until the balloon of the Miller-Abbott tube had issued from it.

The following case illustrates the ineffectiveness of an ileostomy in the treatment of paralytic ileus.

Example 15.—A middle-aged and very obese man had had an acutely inflamed appendix removed. On the fourth post-operative day he began to get distended and to vomit copiously. A Miller-Abbott tube was successfully passed and drew off large quantities of fluid. However, the patient had a slightly elevated temperature and his general condition did not improve. The surgeon reopened the wound, and, on not finding pus, did an ileostomy. On the seventh post-operative day the nursing sister reported a slough in the wound, which turned out to be the bag of the Miller-Abbott tube. The tube was then removed, and the ileostomy commenced to function well.

A further method of treatment of paralytic ileus which is much used is prolonged gastric or duodenal suction with continuous intravenous fluid administration (Wangensteen, 1942 ; Wangensteen, Rea, Smith, and Schwyzer, 1939 ; Penberthy, Noer, and Benson, 1940 ; Bartels, 1943, and many others).

Since most of the gas in the bowel has been shown to be swallowed air, this seems a very logical method of prophylaxis.

Finally, the last method of treatment of paralytic ileus is by use of the Miller-Abbott tube (Abbott and Miller, 1934 ; Ravdin and Abbott, 1940 ; Miller, 1944 ; R. A. Wise, 1938 ; Boon, 1940 ; Penberthy, Noer, and Benson, 1940 ; Lofstrom and Noer, 1940 ; Bennett, 1941 ; Kaplan and Michel, 1941 ; Noer and Johnston, 1942 ; Grimson and Hodge, 1944 ; Glen, 1941 ; Smith, 1945 ; and many others).

Ladd and Gross (1941) stress the value of a high oxygen concentration in a tent in cases of abdominal distension. Probably this helps to maintain the oxygen concentration in the lumen of the affected gut.

Thus there are three schools of treatment :—

1. Stimulative, with enemas, drugs, and cathartics.
2. 'Resting' the bowel with morphine and perhaps heat on the abdomen, turpentine stupes, etc.
3. The aspiration of stomach, duodenum, or the entire intestine.

Each has its advocates. As a result of this clinical study of paralytic ileus, each treatment would seem to be correct at the right period of the disease, but not at another.

II

A study of the treatment of post-operative ileus —and paralytic ileus—logically involves a consideration of the following :—

1. Normal bowel movements.
2. Those drugs and procedures which are used to increase movements.
3. The action on the human small intestine of morphine—a drug frequently used in the treatment of ileus, not only for its action on the muscle of the small intestine, but also for the induction of sleep and relief of pain.
4. Drugs which decrease the motor function of the small intestine and might therefore conduce to post-operative ileus.
5. Drugs used in general treatment which might decrease intestinal movements and conduce to ileus.
6. Coincident or coexisting pathological conditions which might pave the way to a post-operative ileus.

THE NORMAL MOVEMENTS OF THE SMALL INTESTINE

It is necessary fully to understand the normal movements of the small intestine if the treatment of their absence is to be undertaken.

There is an inherent rhythm of bowel muscle even when isolated from a nerve-supply (Thomas and Kuntz, 1926). However, the nerve-supply co-ordinates peristalsis in response to local stimuli and the central nervous system in turn superimposes its control on the reflexes, as exemplified by a book-loving professor who complained of turbulent borborygmi on entering second-hand bookshops.

The irritability and amount of small-bowel movement decreases from above downwards in a gradient (Alvarez, 1928). Small-bowel movements fundamentally consist of rhythmic 'feeler' waves which establish the presence of resistance to contraction (an adequate stimulus as shown by the fact that resistance to contraction can cause pain), and once the presence of the intestinal contents is so determined, a peristaltic wave follows. Bowel movements still occur in denervated bowel (Bayliss and Starling, 1899).

Bowel movements as recorded on a Miller-Abbott tube balloon are irregular and of three main types. The first type occurs five to ten times a minute and is small in amplitude. The second type has the first type superimposed on it and consists of an alteration in tone occurring two or three times a minute. The third type is a big alteration in tone with both the first and the second waves superimposed on it, occurring once every one to three minutes. Personal experience has shown that the presence of the balloon of the Miller-Abbott tube low down in the ileum can be recognized by the type of recording obtained. Frequent type three waves are found in the upper ileum and jejunum where, when the tube is well down in the ileum, types one and two waves are not so marked, and powerful infrequent type three waves are noted (Huidobro, Montero, and Cuevas, 1944; Abbott and Pendergrass, 1936; Forster, 1940).*

DRUGS WHICH ARE USED TO INCREASE THE MOVEMENTS OF THE HUMAN SMALL INTESTINE

As has been pointed out above, one routine treatment of established ileus is the use of drugs which stimulate the movements and increase the motility of the small intestine. The action and use of some of these drugs is described.

Confusion in regard to the Action on the Muscle of the Human Intestine of Drugs and Procedures.—In the literature there exists much doubt in regard to the action of drugs on the human intestine. This is owing to the fact that the effect of drugs or solutions has been tried on isolated intestine; or in animals in which either X-ray, intestinal balloon, or exteriorized loops have been used; or in human beings where X-ray, balloons, or systems of balloons introduced from above through the stomach or from below through an ileostomy have been connected to recording apparatus; or to the fact that observations of various types have been made on exteriorized loops of bowel, or that observations have been made on the abdomen while open at operation. But despite the fact that, for the reasons given above, there is much difference of opinion in regard to the effect of drugs on the movement of the small bowel of animals and man, the action of certain of these drugs seems to be well accepted and to be clear.

Prostigmine.—Stout (1943) advises early use of parasympathetic stimulants as prophylactic treatment against paralytic ileus. He advises 0·5 mg. prostigmine methylsulphate and 1 mg. thiamine hydrochloride every four hours for ten doses as a prophylactic. Marden and Williamson (1939) recommend prostigmine methylsulphate, 1 c.c. of 1–4000 solution as a prophylactic and 1 c.c. of 1–2000 solution to combat developed paralytic ileus.

Ochsner, Gage, and Cutting (1930) are of opinion that physostigmine is the only stimulative drug which has a definite place in the treatment of intestinal atony.

Acetylcholine.—Hartman and Dock (1927) favour the use of choline. Feldberg and Solandt (1942) used isolated rabbits' intestines and found that acetylcholine caused immediate shortening of the isolated gut, but that glucose was necessary for continuance of this excitability of the longitudinal muscles to the drug. Small doses of atropine abolished the stimulating effect of acetylcholine.

* In a case in which there was prolapse of 3 in. of ileum through an ileostomy, repeated observations showed that the papillæ contracted and described rhythmic sigmoid curves indistinguishable from those ascribed to the 'ileocæcal valve'.

Ochsner, Gage, and Cutting (1930) found little effect from acetylcholine on peristalsis in dogs.

Pituitary Extracts, Pitressin, Pituitrin.—Puestow (1942) found that, following the administration of pituitary extract, there was a contrary motility between the small and the large bowel; that when the small bowel was vigorously contracting the colon was inactive, and vice versa. He showed that posterior pituitary extract and pitressin produced powerful contractions of the colon, but diminished motility of the small bowel. Carlson (1930) found that 1 c.c. of pituitary extract in a patient with an ileostomy gave increased peristalsis with issue of gas and fæces. In dogs the result was not so constant. Buirge (1944), who studied the ileocæcal valve in a patient in which it had prolapsed, found that 1 c.c. of posterior pituitary extract increased propulsive and non-propulsive (or mixing) movements in this region and caused gas pains. It produced a segmental contraction of one segment, which was relieved by amyl nitrite.

Pituitary extract and pitressin regularly increased colon motility (Carlson, 1930; Puestow, 1942; Buirge, 1944). Radiologists take advantage of pitressin to rid the bowel of gas shadows, when taking radiographs of the renal tract (Kenning and Lofstrom, 1937). On the isolated small intestine, however, results are more equivocal.

Using isolated dogs' intestines, Gruber and Pipkin (1930) found that high dilutions of pituitary extracts caused an increased tonus, and tonic oscillation in duodenal and ileal strips, whereas high concentrations of the same drug caused loss of muscle tone and disappearance of the rhythmic (or mixing) movements. Huidobro, Montero, and Cuevas (1944) found in normal subjects with a Miller-Abbott tube in position that pitressin increased the spontaneous motility of the small bowel.

Ochsner, Gage, and Cutting (1930) tested the action of pituitary extracts in a series of twenty-nine dogs. They found characteristically a decrease in tone and inhibition of peristaltic movement, and in only two cases an increase in tone. They were of opinion that pituitary extract was not only ineffective, but dangerous, in intestinal atony.

The author made observations on the small bowel with the Miller-Abbott tube in position. In the case reported below the contractions of the small intestine, which up to the time of injection of pituitrin had been painless, became painful and increased.

Example 16.—A man, aged 21 years, had an appendicectomy performed. Three weeks later he began to get colicky abdominal pains and occasional vomiting. A Miller-Abbott tube was passed and relieved his pain and vomiting. He began to eat. Again, one week after the removal of the tube, he began to vomit and to become distended. At this stage he had visible peristalsis. A Miller-Abbott tube was again passed. A second operation was performed and many adhesions caused by plastic peritonitis were divided. During his recovery the Miller-Abbott tube, which was in position, was connected to a recording tambour. While it was in the lower ileum, doses of pituitrin of 1 c.c. were given on two occasions. Within twenty minutes of the giving of the intramuscular injection the patient complained of colicky central abdominal pains which coincided with increased peristaltic waves as shown on the recording tambour. Luminal, in 1-gr. doses, decreased the peristaltic activity for some hours.

These diverse experimental findings in human beings and animals may perhaps be reconciled thus: If the colon is intact and pituitrin stimulates the motility, then it may later increase the small-bowel motility by a reflex. (*See* later.)

Cathartics.—Stimulant treatment with cathartics increases the movements of the normal small bowel (Oppenheimer and Mann, 1941).

Castor oil causes increased amplitude and rate of peristalsis in exteriorized loops of bowel in dogs. *Magnesium sulphate* does not increase the rate of movements or tone of the small bowel; it has only a mechanical action. *Cascara* does not change the rate of movement, but occasionally increases the response to local stimulus.

In established paralytic ileus cathartics have no effect whatever. If cathartics 'work', the patient has not got paralytic ileus. This case of paralytic ileus—one of many—demonstrates it.

Example 17.—A middle-aged woman had had a hysterectomy performed. On the second day she was given an enema without result; then cascara and paraffin and magnesium sulphate without either getting a result or causing colicky pains. Next day more cascara and another enema were given, again without result. When seen on the fourth day she was very distended, and on auscultation of her abdomen occasional sounds were heard. She recovered after $\frac{1}{6}$ gr. morphine had been given four-hourly for three days. Her bowels then opened with an enema.

Therefore, though cathartics may at times slightly increase the movements of the small bowel for a short while, they should have no place in pre- and immediate post-operative treatment of patients undergoing surgical operations. Their use before operation is followed by a period in which there is less bowel motility and a decreased response to local stimulants. Furthermore, cathartics irritate the bowel, increase the fluid contents of the bowel, and prevent absorption of nutritive material from it. Following purgation, the bowel does not react well to drugs, and there is an increased growth of bacteria within its lumen, and if continued there is increased gas and fluid in the lumen, a condition predisposing to the development of paralytic ileus (Alvarez, 1918; and Alvarez and Taylor, 1917–18).

Apomorphine.—According to experiments on dogs, apomorphine causes peristaltic waves in the small intestine associated with attempts to vomit, which are followed by a decrease in the height of the contractions and in muscle tone, lasting from twenty to sixty minutes (Slaughter and Gross, 1938).

Vitamins.—Crandell, Chesley, Hansen, and Dunbar (1939) found that, following the giving of thiamine and riboflavine and nicotinic acid to dogs and human beings with functional digestive disorders, some cases exhibited an intestinal hyper-motility.

It is the author's experience that the use of vitamin B complex has been most effective as a help in dealing with habitual constipation, but it has also been noticed on numerous occasions that patients to whom these drugs have been given are conscious of painful intestinal motility (umbilical pain) while they are taking them.

Some patients who have been given ascorbic acid also complain of colicky abdominal pains. This also may be due to hypermotility, for Haag and Taliaferro (1940) found that, at least in the colon (isolated from a guinea-pig), ascorbic acid caused greatly increased tone.

Bran.—No change in the movements or evacuation of the small intestine was found by Fantus, Kopstein, and Schmidt (1940) and Streicher and Quirk (1943) in making X-ray studies of bowel motility in the normal individual.

Bile and Bile-salts.—Haney, Roley, and Cole (1939) found that bile or bile-salts increased the rate of propulsion through isolated loops of dogs' intestines, hence the importance of giving bile-salts in paralytic ileus.

Hypertonic Saline into Bowel Lumen.—Bayliss and Starling (1899) mention that hypertonic saline solutions injected into the lumen of the gut are followed by very vigorous peristalsis.

PROCEDURES WHICH ARE USED TO STIMULATE THE MOVEMENTS OF THE SMALL INTESTINE

Intravenous Hypertonic Salt Solution.—Among the stimulant forms of treatment must be mentioned the intravenous infusion of hypertonic saline solution as advocated by Ochsner, Gage, and Cutting (1933). From 15 to 20 c.c. of 20 per cent sodium chloride solution, injected intravenously, has been found to increase small-bowel motility (Ochsner, Gage, and Cutting, 1930; Kleitch and Puestow, 1939; Hughson and Scarff, 1924; Quigley, Highstone, and Ivy, 1934). Kleitch and Puestow (1939) found that physiological saline given intravenously for twenty minutes gave a sustained rise in the frequency of contraction and also in the secretion of the small intestine of dogs. Dextrose solution (10 per cent) did not give much change, but physiological saline with 10 per cent dextrose in it definitely increased motility. They found that insulin decreased the motility. Their observation confirms that of Feldberg and Solandt (1942) that the action of nicotine on the intestine depends on the presence of glucose in adequate supply.

Spinal and Splanchnic Anæsthesia.—There is little doubt that where small-bowel motility is arrested or diminished, spinal and splanchnic anæsthesia causes an increase; at any rate, during the time the anæsthesia is active (Wagner, 1919; David and Loring, 1930; and Novikov, 1940). When spinal anæsthesia was used on patients who had morphine as a pre-operative treatment, little effect on the motility of the small intestine from spinal anæsthesia was found by Helm and Ingelfinger (1944). The following case illustrates this :—

Example 18.—A woman had an acute obstruction of the small intestine. A Miller-Abbott tube was passed for the two hours during which she was being prepared for the theatre. At operation the tip of the tube was found to be in the intestine about 2 ft. from the pylorus. Before operation little or no motility could be observed on the balloon. As soon as the spinal anæsthetic was given, waves of motility were recorded on the tambour, and at operation they were seen.

Food by Mouth.—The gastro-ileal reflex is physiologically well known. It can be observed on any patient with an ileostomy. Following the taking of food, the ileostomy acts with increased vigour.

Colonic Enemas and Wash-outs.—Repeated observations on a man who had an acting ileostomy at the same time as he had a colon isolated from the fæcal stream as a preliminary to its removal (*Fig.* 228 shows this arrangement) showed that whenever

FIG. 228.—To show the arrangement of loops of bowel as a preliminary to subtotal colectomy for ulcerative colitis, the ileum being eventually joined to the rectum by clamp. 1, End of functioning ileum; 2, End of functioning colon; 3, Functioning ileostomy; 4, Cut end of rectum.

either the colon or the rectum was washed out, or an enema given, within five or ten minutes there was a most vigorous action of the ileostomy. At times this action was so vigorous that jets of chyme were shot several feet into the air.

These observations were repeated and confirmed on a woman who also had an isolated colon as a preliminary to colectomy (*Fig.* 229).

Thus enemas and bowel wash-outs must be regarded as *the most effective stimulative treatment* to a small bowel capable of response.

Heat to the Abdomen.—This time-honoured method of treating paralytic ileus, which is supposed to increase the motor function of the bowel, and particularly the pendulum movement, was found to have no effect. In three patients in whom heat was applied to the abdomen while the recording Miller-Abbott tube was in position, no change in the motility of the intestine was observed.

Example 19.—This patient had a tube in position following operation for division of adhesions which were causing intestinal obstruction with visible peristalsis. On two occasions a radiant heat cradle was put on the naked abdomen for periods of from half an hour to one and a half hours without altering the character or strength or frequency of the bowel movement as recorded on the Miller-Abbott tambour.

Example 20.—The patient had a Miller-Abbott tube both before and after an operation for division of multiple adhesions of the small bowel. On two occasions hot bottles applied to the abdomen did not in any way alter the character or frequency of intestinal contractions.

Example 21.—A young woman had an ileostomy as a preliminary to colectomy. A balloon was introduced into the ileostomy for about 4 in. Heat was applied to the abdomen by hot-water bottles for twenty minutes, and also a turpentine stupe at the same time, without affecting the bowel motility (*see Fig.* 229.)

twenty to forty minutes after the injection, but for some hours later the rate of propulsion was slower than normal. Atropine was found to counteract the effect of the morphine.

Using similar preparations Gruber, Greene, Drayer, and Crawford (1930) found a rise of tone following injections of morphine.

Determining by X rays the rate of propulsion in Thiery-Vella loops in dogs, Quigley, Highstone, and Ivy (1934) found, as the result of morphine injection, an increased rate of propulsion in the

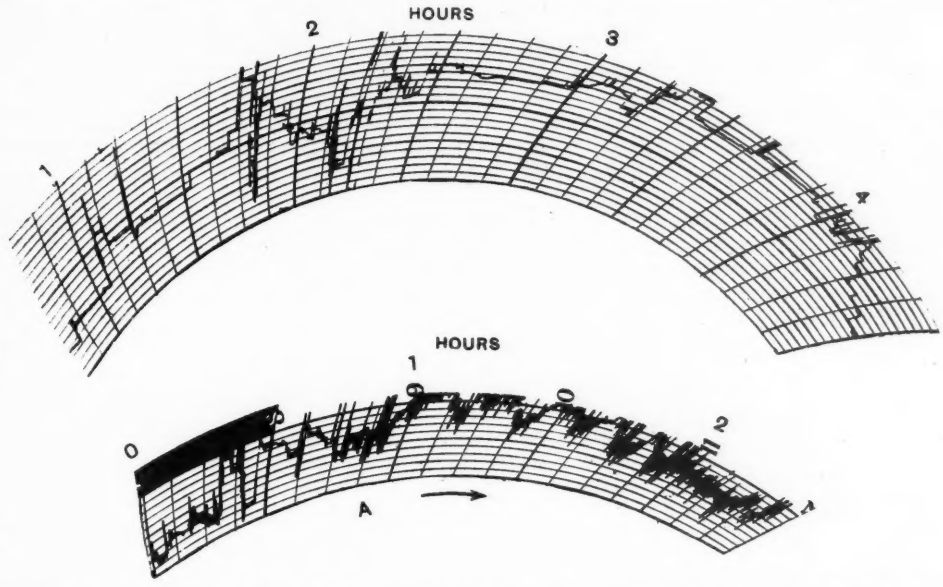


FIG. 229.—*Example* 42. *a*, A record of four and threequarter hours taken immediately after operation when little or no bowel movement was present, showing variations in the record produced by coughing, vomiting, gasping, etc. *b*, A record for two and a half hours showing the development of movements 10 hours after introduction of the tube through jejunostomy. First movements shown in *Fig.* 238. From the mark A the record is typical of fairly normal jejunal movements. (Cf. *Fig.* 234, where the movements of the one-and-three-quarter-hour period to the mark C are typical of normal lower ileal movements.)

It has also been found by McLoughlin, Mann, and Krusen (1941) that short-wave diathermy does not affect intestinal motility.

Turpentine Stupes on the Abdomen.—This also time-honoured treatment was used in *Example* 21 mentioned above, with no effect either clinically or on the bowel movement as shown by the recording tambour (*Fig.* 229).

THE ACTION OF MORPHINE ON THE HUMAN SMALL INTESTINE

With regard to the effect of morphine on the human small intestine, some confusion exists because, as has been pointed out, workers have sought information in this respect in so many different ways. Thus, if the morphine method of treating ileus is to be intelligently carried out, its mode of action must be studied and, if possible, clarified.

Using isolated strips of intestine, Gruber and Pipkin (1930) found that morphine increased the tonus and in some cases the height of rhythmic contraction of the muscle.

Using experimental animals, Kanan (1937) placed balloons in Thiery-Vella loops in the ileum and measured propulsive motility, and found that morphine sulphate increased propulsion for from ileum lasting for forty minutes, then a decrease for some hours. In the loops of the duodenum the initial increase of propulsive activity was greater than in the ileum.

Plant and Miller (1926), using non-anæsthetized dogs, also found an immediate increase of frequency in the amplitude of peristaltic waves, but, when this passed off, the tone still remained above normal.

The following observations on man when contrasted with that of the animal intestine show some discrepancies :—

Plant and Miller (1926) took tracings from the ileum of a man with an ileostomy and found that $\frac{1}{6}$ gr. morphine increased the number of waves and the tone, but decreased the amplitude of peristaltic waves. They saw similar changes in two men with thin-walled herniæ when, after the giving of $\frac{1}{4}$ gr. morphine hypodermically, contractions were noted and an increase of the frequency of waves passing over the bowel was perceived. The appearance did not return to normal for one and a half hours after injection of morphine. They found similar effects by using heroin and codeine.

Forster (1940) studied movements of the circular and longitudinal muscles separately by means of recording instruments attached by stitches to the

exteriorized ileum in man. He found that $\frac{1}{8}$ gr. morphine increased the frequency of the mixing waves and the peristaltic waves, and that a second injection of $\frac{1}{8}$ gr. twenty to thirty minutes later decreased the frequency of the mixing waves or pendulum movements, but increased the tone of both muscle coats.

Huidobro, Montero, and Cuevas (1944), recording from the balloon of the Miller-Abbott tube in normal subjects, found that morphine decreased the motor activities in the duodenum and ileum. In large doses it caused movement to disappear with slightly increased tonus.

Abbott and Pendergrass (1936), using a Miller-Abbott tube, found that morphine produced stimulation in the upper part of the small bowel for a short period, followed by depression, but there might be no change at all in the motility of the ileum. There was duodenal spasm for two minutes following the injection and then relaxation lasting up to three hours. Barium conglomerated in the ileum and filled loops, giving the appearance of a string of sausages. Disordered and non-propulsive movements persisted for three hours after injection. The effects of morphine on the gastro-intestinal tract lasted for up to three days, the ileum emptying more slowly for twenty-four hours.

Plant and Miller (1926) state that the primary effect of morphine on the small intestine is stimulation of muscular activity and tone, while contractions remained unaltered or even slightly decreased.

Myers and Davidson (1938), reviewing the literature, came to the conclusion that the constipation of morphine was due to pyloric obstruction and consequent delay of stomach emptying.

Puestow (1942) states that morphine constantly stimulates the small bowel, but by inhibiting colonic contraction may be a factor in causing post-operative distension due to gas distension.

The author studied three cases in which tracings and observations were made on patients with the Miller-Abbott tube and recording tambour. In these cases no gross difference could be observed in the contractions after $\frac{1}{6}$ gr. morphine had been given intramuscularly. But in one case, on five occasions when a balloon was introduced through an ileostomy (*Fig.* 230) tone was increased, mixing movements were more frequent, and peristaltic movements were decreased.

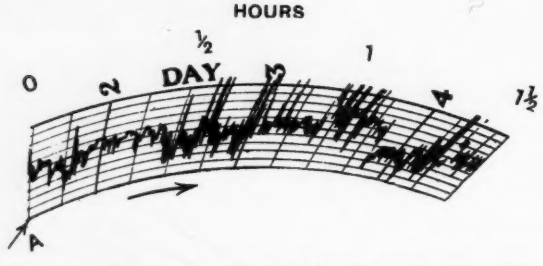


FIG. 230.—*Example* 42. To show the effect on bowel motility of an injection of $\frac{1}{8}$ gr. morphine given at A.

The treatment of paralytic ileus by injections of morphine has therefore a sound physiological basis, not because it rests the bowel but because it increases the number of small mixing waves and raises the muscle tone. Both these actions tend to prevent the onset of an 'irreversible' stage of ileus. This latter is the stage of ileus which occurs when distension has increased to such intensity that (*a*) it presses on the veins and so causes a vicious circle in which decreased gas absorption resulting from the venous pressure leads to more distension and thus further venous pressure (Dragstedt, Lang, and Millett in 1929 showed that distension interfered most with blood-supply in the duodenum and least in the large bowel); and (*b*) it causes of itself an actual mechanical obstruction by a kinking of the ends of the loops (*see Figs.* 226, 227).

Treatment with morphine will prevent this intensity of distension and therefore facilitates the absorption of gas by increasing the muscle tone and by increasing the number of small mixing waves.

To sum up, the results of the investigation of morphine seem to show that there is agreement that morphine: (1) delays the passage of food through the intestine and therefore causes constipation; (2) causes duodenal spasms for a short while, followed by relaxation; (3) decreases peristaltic movement of the duodenum and ileum, but increases (*a*) the tone, and (*b*) the frequency, of the small mixing waves.

DRUGS WHICH DECREASE THE MOTOR ACTIVITY OF THE HUMAN SMALL INTESTINE

Drugs which depress the motor activity of the human small intestine have a bearing on the treatment of ileus, for their use in post-operative treatmen may have an influence in the production of post operative paralytic ileus.

Atropine.—Atropine given in normal doses causes inhibition of contraction and loss of tone of the small bowel (Forster, 1940; Huidobro, Montero and Cuevas, 1944; Gruber, Greene, Drayer, and Crawford, 1930; Kanan, 1937).

Trasentin causes a decrease in small-bowel movement, especially when given through the Miller-Abbott tube. Local anæsthetics given through a Miller-Abbott tube also decrease the bowel movement (Crohn, Olson, and Necheles, 1944).

In only one of three cases was trasentin effective in relieving spasms caused by intestinal colic resulting from adhesions due to plastic peritonitis.

The author found no effect by giving trasentin by mouth in a case of chronic small-bowel obstruction when the Miller tube had been passed pre-operatively and the balloon was in the lower ileum.

Hyoscine has an action similar to, but weaker than, atropine (Gruber, Greene, Drayer, and Crawford, 1930).

Adrenaline and Neosynephrin slow the contractions and decrease the tone of the intestine in dogs (Youmans, Aumann, and Haney, 1939).

Amyl Nitrite decreases both intestinal movement and tone. In the following case the effect of amyl nitrite on intestinal movement is seen.

Example 22.—A patient came into hospital with intestinal obstruction. She had had nine previous operations for adhesions and obstruction following a hysterectomy operation. Before being operated upon there was visible peristalsis in the abdomen and colicky pains, both of

which were abolished by the giving of amyl nitrite. A Miller-Abbott tube was passed before operation, which emptied her intestine and stopped her pain. At operation the collapsed bowel and the site of obstruction, threaded on the Miller-Abbott tube, were easily found.

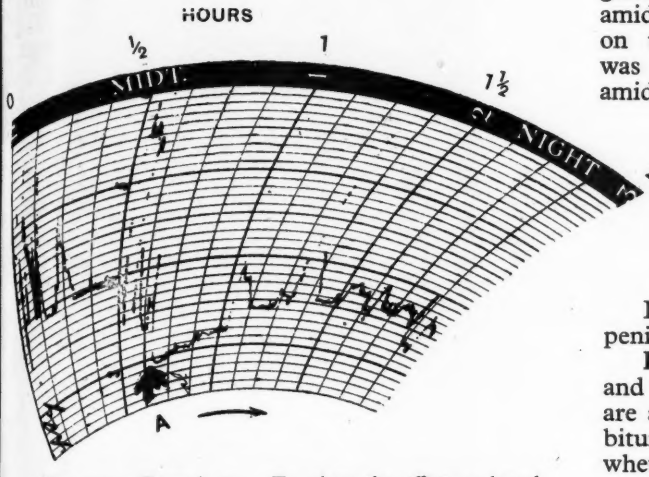


FIG. 231.—*Example* 25. To show the effect on bowel motility of 2½ gr. of sodium luminal given intravenously at A. This dose was enough to make the patient feel sleepy, but not to put him to sleep without morphine given later.

Huidobro, Montero, and Cuevas (1944) also report decreased intestinal movement and tone after the use of amyl nitrite.

Sulphonamides.—The author has studied the action of sulphonamides on the human intestine in a number of cases. He has found no appreciable difference in small-bowel motility when sulphonamides are given.

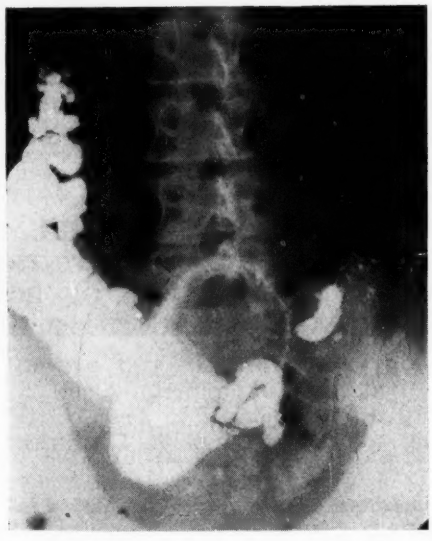


FIG. 232.—Radiograph four hours after taking barium by mouth.

The following is an example from a number of cases observed :—

Example 23.—A woman developed paralytic ileus, with a high temperature, following a gynæcological operation. She was given sulphadiazine, 4 g. immediately and 1 g. every four hours for four days. All this time she had a Miller-Abbott tube in her ileum and strong peristaltic contractions continued.

Pick, Brooks, and Unna (1944) found that in guinea-pigs with isolated intestinal loops sulphonamides inhibited the contracting effect of nicotine on the isolated intestine. Sodium sulphadiazine was not so effective as sulphathiazole, sulphanilamide, or sulphamerazine.

DRUGS FREELY USED IN THE GENERAL TREATMENT OF ILEUS WHICH MAY DECREASE INTESTINAL MOVEMENTS AND CONDUCE TO A POST-OPERATIVE ILEUS

In this category are the barbiturates, aspirin, and penicillin.

Barbiturates.—Work by Professor R. D. Wright and the fact that epileptic patients on luminal therapy are always constipated suggested an inquiry if barbiturates affect intestinal movements, and if so whether they might produce post-operative ileus.

Barbiturates were given to three patients in whom the Miller-Abbott balloon with an attached tambour was in the small intestine. Two-grain doses of luminal, given each night, caused a significant decrease in the amplitude of contraction for five or six hours. Subsequently no marked difference in muscle tone could be detected.

Example 24.—In this patient a balloon was introduced through an ileostomy. A 3-gr. dose of seconal was given and within half an hour the tambour recorded an increase of tone, and then almost complete cessation.

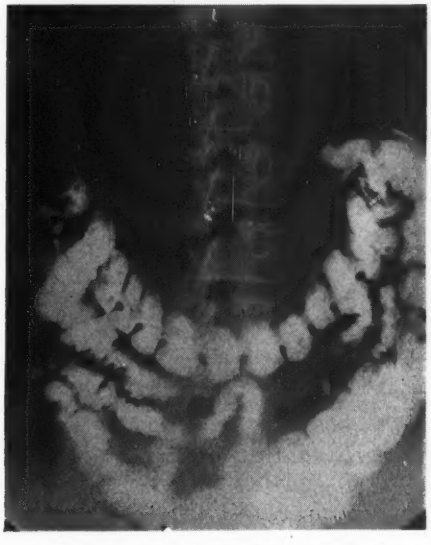


FIG. 233.—Radiograph after the taking of barium and 1 gr. of luminal. The colon is still full from the day before.

Example 25.—In this case an ileostomy had been performed as a preliminary to removal of the colon. A post-operative ileus ensued. A Miller-Abbott tube had been introduced through a jejunostomy because the patient persistently pulled it out of his nose. On four occasions injections of 2½ gr. of sodium luminal were

given intravenously; this was enough to make him sleepy. The tambour recorded a depression of motility which lasted several hours (*Fig.* 231).

An X-ray study of the effect of luminal on bowel movement was undertaken on a middle-aged volunteer.

Example 26.—Observations were made under identical conditions at the same time of the day, viz., four hours after the giving of four tablespoonfuls of barium by mouth. In the control experiment the tail of the barium column at the end of four hours was 6 in. from the ileo-cæcal junction. When 1 gr. of luminal was given to the same subject under the same conditions, the subject complained of much sleepiness, and the tail of the barium column was at least 2 ft. and possibly 3 ft., from the ileocæcal junction (*Figs.* 232, 233).

There was thus a retardation of the flow of barium through the small intestine following the administration of the luminal.

Using isolated intestine of rats or rabbits, Shaw (1942) found that luminal, dial, seconal, etc., caused a decrease in tone and sometimes a cessation of motility.

Florey, Wright, and Jennings (1941) noticed that the secretion of mucus in the colon was decreased by barbiturates.

It would appear, therefore, that barbiturates should be given with caution in cases in which there is

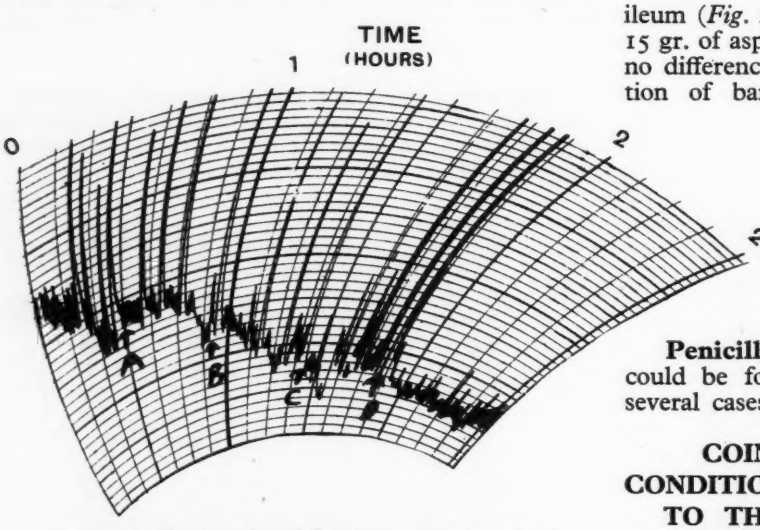


FIG. 234.—To show the effect of turpentine stupes (B) (applied for twenty minutes), heat to the abdomen (A) (applied for twenty minutes), the wash-out (with half a pint of saline) of the disconnected rectum in which the rectum was joined to the ileum (C), and of 10 gr. of aspirin (D), on bowel movements as recorded from a balloon introduced through a prolapsing ileostomy from the disconnected terminal ileum. (The ileumw as connected to the rectum as a preliminary for colectomy and the distal disconnected ileum and sigmoid colon were brought out on to the abdominal surface.)

a possibility of paralytic ileus. They certainly should not be given in cases of established paralytic ileus as they decrease peristalsis and tone, and consequently as a result of the increased distension, might just change a developed paralytic ileus which is in a 'reversible' stage into one which is 'irreversible.'

Aspirin.—In a middle-aged woman with a recording Miller-Abbott tube in position, who had had a resection for a carcinoma of the ileum, 10 gr. of aspirin almost caused cessation of ileal movement. In a woman of 35 years who had an ileostomy for ulcerative colitis, 10 gr. of aspirin abolished the larger occasional 'peristaltic' movements of the

FIG. 235.—Radiograph four hours after barium by mouth plus 15 gr. of aspirin, under the same conditions as *Fig.* 234.

ileum (*Fig.* 234). In a normal middle-aged subject, 15 gr. of aspirin (given with barium emulsion) made no difference, compared with a control administration of barium without aspirin under identical conditions (*Fig.* 235). The dose of aspirin, 15 gr., was enough to make the patient "sleepy".

It thus appears that the rate of propulsion of the content of the small bowel is not materially altered by aspirin—though occasionally decrease of intestinal movement followed its exhibition.

Penicillin.—No effect on intestinal movement could be found with large doses of penicillin in several cases.

COINCIDENT PATHOLOGICAL CONDITIONS WHICH MAY CONTRIBUTE TO THE OCCURRENCE OF POST-OPERATIVE PARALYTIC ILEUS

Hypoproteinæmia.—In two patients with a Miller-Abbott tube in position, the author was able to record definite increase in small-bowel motility following serum transfusions. Their case records are as follows:—

Example 27.—A Miller-Abbott tube had been introduced for a post-operative ileus. No progress was made after it was 4 or 5 in. past the pylorus. Two pints of serum were given. Movements increased in amplitude and frequency, and the tube began to move and fluid was aspirated.

Example 28.—A woman who had had many previous operations for intestinal obstruction had a Miller-Abbott tube passed prior to operation. At operation it was found to be proximal to the obstructed segment of bowel. Twenty-four hours after operation occasional movements

of the bowel were being observed. Following the administration of a pint of intravenous serum, these quickly increased in frequency and amplitude.

That hypoproteinæmia is a potent cause of depression of small-bowel motility has been reported by Barden, Thompson, Ravdin, and Frank, 1938; Leigh, 1942; John Devine, 1938; Ravdin, Stengel, and Prushankin, 1940.

recommences when the packing is removed. (*See Example* 3.)

The cessation of bowel movement resulting from intraperitoneal irritation does not occur if the splanchnic nerves are cut, as they are part of the reflex arc through which inhibition of bowel movement occurs (Douglass and Mann, 1941), and the interference with this arc is the rationale for

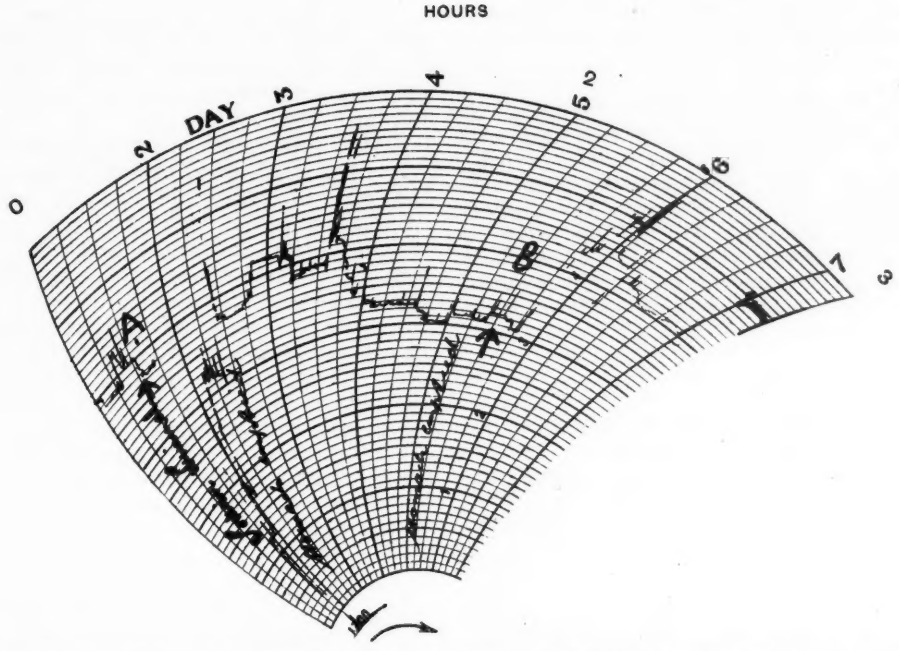


FIG. 236.—*Example* 42. To show the momentary complete loss of tone after giving 2½ gr. of luminal intravenously at A, a dose which did not render the patient unconscious. It also illustrates the depression of peristalsis and tone resultant upon overdistension of the stomach and the effect of relief of this by removal of 100 oz. of fluid through a stomach tube at B.

Distension of the Upper Part of the Urinary Tract or of the Bladder.—In cases of urinary tract irritation, Allen and Jamison (1940) found increased bowel distension, indicating a degree of ileus, and confirmed by radiography.

Svien and Mann (1943) showed that distension of the urinary bladder caused inhibition of movements of the lower ileum, but distension of the upper urinary tract caused ileus of the whole of the small intestine.

It would appear that the type of patient who has urinary retention following operation has also a tendency to ileus. On the basis that distension of the bladder or pelvis of the kidney causes ileus, it is suggested that unrelieved post-operative retention of urine might contribute to or even be the origin of paralytic ileus—this is an important practical point in post-operative treatment.

Intraperitoneal Procedures: Intraperitoneal Irritation.—Intraperitoneal procedures or intraperitoneal irritants cause prolonged arrest of small-bowel movement (Douglass and Mann, 1941; Wakim and Mann, 1943).

Arrest of small-bowel movement for a varying period results from intraperitoneal procedures after every abdominal operation, whether a general or local anæsthetic is used; e.g., packing introduced into the abdominal cavity may cause prolonged cessation of small-bowel movement which will only

using splanchnic or spinal anæsthesia in the treatment of 'true' paralytic ileus.

Over-distension of the Stomach.—The author has found that over-distension of the stomach inhibits intestinal movement.

This observation was made possible because jejunostomies had to be done on two desperate cases of paralytic ileus, too ill and unco-operative for the tube to be passed by mouth. Both had a recording Miller-Abbott tube passed into the ileum through the jejunostomy. In each case the stomach and upper part of the jejunum, unrelieved by the Miller-Abbott tube, remained greatly distended. In each case the tambour recording movements of the small intestine occasionally showed a complete absence of peristalsis. And in both of these cases, following aspiration of varying amounts of brownish fluid up to 100 ounces through a separate Rehfuss tube introduced through the mouth into the stomach, the result was that the tambour recorded the return of peristalsis.

The details of these cases are as follows:—

Example 29.—A young woman had had a paralytic ileus following a gynæcological operation. This in turn was followed by peritonitis, and finally, a fortnight after the operation, by organic small-bowel obstruction. After the Miller-Abbott tube had been passed twice by mouth, it could not be passed on a third occasion. It was then passed through a jejunostomy made under local

anæsthesia. Eight hours afterwards the first small-bowel movements were recorded. On several occasions peristalsis was observed to stop, and on each occasion aspiration of the stomach drew off a large quantity of fluid, and within half an hour peristaltic movements returned.

Example 30.—The Miller-Abbott tube was in the ileum, having been introduced through a jejunostomy, and cessation of peristalsis was on several occasions associated with epigastric fullness. A stomach tube was then used to draw off up to 128 oz. of fluid. Immediately peristalsis recommenced (*Figs.* 227, 236). This was repeated on at least five occasions.

This reflex, resulting from gastric distension, is physiologically reasonable, since distension of an isolated loop of small bowel and of the urinary bladder has already been shown to cause inhibition of small-bowel peristalsis, and therefore it is important to prevent over-distension of the stomach following operations, and to anticipate any tendency to acute gastric dilatation by promptly emptying the stomach by a Rehfuss tube before inhibition of intestinal movements can occur.*

III

THE DEVELOPMENT OF A CONCEPT OF PARALYTIC ILEUS

In a fully developed paralytic ileus auscultation of the abdomen reveals no sounds and a recording tambour in the intestine shows no movements.

In this clinical study it was found that in a person in good health intestinal sounds (borborygmi) were always heard in the abdomen. They were usually most pronounced after a meal, and had a certain regularity. Abdominal auscultation made on a number of patients following routine operations, showed that the first sounds were heard between four and eight hours after the operation. It also disclosed the interesting fact that the first sounds are irregular, turbulent, and often accompanied by pain ; at first they were infrequent and later become almost continuous, quite unlike those heard in the normal abdomen.

The cause of these first sounds following operation was investigated by the use of the Miller-Abbott tube with recording tambour, as is seen in the following case.

Example 31.—A middle-aged woman had had nine previous operations for intestinal obstruction caused by a constant recurrence of adhesions which originated as a sequel of a hysterectomy. An acute supervening obstruction with distension and visible peristalsis made a tenth operation necessary. The Miller-Abbott tube was passed and relieved the distension and made the subsequent operation technically easy as no distended bowel was encountered, but the tip of the tube was found proximal to the area of obstruction. The bowel was dissected free and wrapped in omentum. Following the operation no movement occurred for eight hours. The recording tambour on the Miller-Abbott tube balloon then began to show several movements at intervals of half to one hour—each movement being accompanied by colicky central abdominal pain. These powerful, but occasional, contractions then changed in about twenty hours to frequent irregular contractions, and after a further twelve hours the peristalsis recorded was indistinguishable from normal.

From these observations it would appear that, at the commencement of intestinal movements, after the normal period of 'physiological' or 'active' ileus following the opening of the abdominal cavity, there is a stage when there is no movement; then one when there is occasional irregular movement; later a stage in which there is more frequent but disordered movements, until finally the bowel recovers and its movements become ordered and normal.

After operations patients usually complain of 'gas pains'. Normal peristalsis is painless, therefore the cause of these pains must be some abnormality in internal movement. And the most likely one, judging from abdominal auscultations which the author has carried out and also from the tambour records, is inco-ordination.

If pain is caused by peristaltic contraction, then the only conceivable cause is that the bowel content cannot escape into the next segment, because of lack of the usual co-ordination, namely, contraction above followed by relaxation below. Wangensteen (1942) thinks that gas pains are the result of contraction of dilated loops on to their contents.

The author found clinically that, given in the first stage, when no movements can be heard, enemas are quite ineffective. They do neither good nor harm: they cause the patient no pain, being either retained or returned without force.

He found that in the second stage, or 'stage of disordered movement', stimulation by enemas, pituitrin, pitressin, eserine, or by other measures causes more distension and increased pain and discomfort for the patient. There is usually some passage of flatus, with little fæcal result. In other words, it would appear that the stimulation caused painful bowel movements. Since normal peristalsis is painless, it is reasonable to assume that the pain was a result of abnormal peristalsis. It is also reasonable to assume further, that, since abnormal peristalsis is disordered and inco-ordinated movement, stimulation by giving pituitrin or enemas increases small-bowel movement without changing the character of the peristalsis present in this second stage. Thus, stimulation in this stage may increase the paralytic ileus. That this assumption is correct is shown by the fact that ileostomy wash-outs given in this second stage to two patients caused 'gas pains' almost immediately, with increased distension.

The following case record shows how stimulation can turn a condition of ileus into one of paralytic ileus :—

Example 32.—A middle-aged woman had an operation for band obstruction of the small intestine, which occurred as a complication of a resection of a volvulus of the sigmoid colon. The band had almost completely obliterated the lumen of the small bowel. Greatly distended proximal loops of small intestine were found. The band was removed and the post-operative convalescence was normal until an enema was ordered on the third day. The enema caused abdominal pains and yielded a poor result. The surgeon then ordered a

* It must be understood that the apparent reflex above mentioned was noted in abnormal intestine under abnormal conditions and with a balloon further upsetting the bowel.

high bowel wash-out. The result of this was an immediate and most marked distension within an hour. The distension was so intense that it interfered with respiration. Later vomiting began. The patient was then given $\frac{1}{6}$ gr. morphine four-hourly, with continuous duodenal suction by a Rehfuss tube. A day later distension was noticeably less and occasional movements could now be heard on auscultation of the abdomen. Within three days the distension was completely relieved, the tube was removed, and convalescence from then on was uneventful.

Incidentally, this case shows how an early stage of paralytic ileus can be treated by morphine to forestall the advent of an irreversible stage which would require the use of the Miller-Abbott tube.

Other cases are here quoted to show how stimulative treatment in the second stage of bowel movement following operation can bring about a paralytic ileus :—

Example 33.—In a patient, following an interval appendicectomy, colicky central abdominal pains unrelieved by morphine set in on the evening of the second day. An enema was given and retained without distress to the patient. The pain was unrelieved and a second enema of milk and treacle with 1 c.c. of pituitrin was given, again without relief of pain, and the enema was also retained. Following this enema, distension became greater. The next morning the pain had ceased but the distension was intense and was interfering with respiration. The patient was then treated with injections of morphine, and the bowels were opened, by an enema, on the sixth post-operative day.

Example 34.—Following a gynæcological operation a patient complained of slight colicky abdominal pains on the second day. She was given an enema without relief and without result. On the following day she was given pituitrin and an enema, with slight flatus result and slight relief of the colicky pain. Following the enema, however, the distension became more marked. The next day she was given another enema and more pituitrin, again with only a small result and increased distension. By the fifth day she had a well-developed paralytic ileus and a Miller-Abbott tube had to be passed.

Example 35.—A middle-aged woman had had a vaginal hysterectomy performed and two days after operation became distended and complained of abdominal pain. On several occasions she was given pituitrin and enemas, each time with slight relief of discomfort, but more distension. Cascara and paraffin were given to her without result or discomfort. When, on the fourth day, she was given $\frac{1}{6}$ gr. morphine four-hourly, her distension lessened day by day until her bowels were opened on the sixth post-operative day.

The 'third stage' of recovery from paralytic ileus, or the recovery of movement following operation, is when once more peristalsis is co-ordinated, regular, and painless. In this stage enemas, pituitrin, bowel wash-outs, etc., produce a result with relief of any symptoms.

Thus, the writer puts forward the concept that there are three stages of ileus : (1) The stage of no movement (paralytic) ; (2) A stage of disordered and unco-ordinated movement ; (3) A stage when movement is being co-ordinated once more.

It is suggested that this concept of ileus should serve as a basis for prophylactic and rational treatment of paralytic ileus.

THE TREATMENT OF PARALYTIC ILEUS

From the results of these experimental inquiries and of these clinical studies the following basic principles emerge in regard to the treatment of paralytic ileus :—

1. That in an ileus or bowel obstruction the chief cause of death is the result of increased permeability of the capillaries of the gut wall, the viability of which has been impaired : (*a*) from the action of bacteria from within, and (*b*) from interference with the circulation by distension. Therefore therapy must be aimed at a reduction of internal pressure to circumvent the impairment of the capillaries ; or at an increase of tone in the circulation to prevent an increase of the distension to the point of irreversibility.

2. That 'true' paralytic (idiopathic) ileus is rare. Many cases which were regarded as idiopathic, were really caused by areas of peritonitis or pockets of infection, toxic or traumatic.

Paralytic ileus caused by infection was often a semi-mechanical obstruction caused by a segment of small bowel which had become paralysed and incapable of passing on intestinal contents because it lay in a focus of pelvic peritonitis or formed part of the wall of a pelvic abscess.

A history of colic, the result of auscultation of the abdomen, and X-ray examination all help to make the important differential diagnosis of this condition from a true mechanical obstruction.

3. That there should be a careful consideration early in every case of paralytic ileus, as to whether or not a mechanical obstruction is present. The paralytic ileus can be a final stage of a neglected mechanical obstruction. If this is the case, and it is not promptly relieved, no exhibition of drugs, intestinal intubation, or other treatment will save the patient's life.

A BASIS OF RATIONAL TREATMENT

Prophylaxis.—As the result of this clinical study and concept of paralytic ileus, the following treatment is advocated :—

1. No cathartics should be given before operation.

2. Gentleness of manipulation during operation ; the avoidance of peritoneal contamination with pus, particularly in cases of localized appendicular abscess ; as few surfaces denuded of peritoneum as possible should be left which might give rise to a reflex inhibition of intestinal movements, and packing should never be left in the peritoneal cavity.

3. In the first twelve hours following an operation, fluid should not be 'forced', since the stomach at this stage usually empties very slowly, and the intestines do not move to enable absorption to occur. Sipping of fluid at this stage must lead to swallowing of much air, which has been shown to be one of the main constituents of the 'gas' in ileus.

4. In the immediate post-operative phase the giving of atropine and barbiturates should be kept at a minimum, and morphine should be substituted for these drugs.

5. Stimulation by enemas, pituitrin, eserine, etc., should not be carried out during the first or second stages of the 'active' ileus which usually follows

any abdominal operation. Such treatment should be given only when there is some certainty that the third stage has been reached. This means that the hearing of abdominal borborygmi is not an indication for immediate stimulation. If it is accepted that the first returning bowel movements are irregular and unco-ordinated, it is unwise to give enemas or pituitrin until all danger of this stage has passed; usually this is after the third day. Since no harm results from leaving the bowels unopened for five, six, seven, or more days, it is in the author's opinion safe to give an enema any time after the fourth day, when the patient feels as if his bowels need opening. In many cases he has found that after the fourth day the bowels open normally without the aid of an enema or cathartics.

Where there is a possibility of the development of any grade of peritonitis and/or ileus, it would appear to be a good practice to put the patient on $\frac{1}{6}$–$\frac{1}{8}$ gr. of morphine four-hourly for its action in increasing the tone and the mixing movements while decreasing the propulsive bowel movements, and to administer penicillin and sulphonamides rather than enemas.

The following case histories, chosen from a number, illustrate the effect of repeated injections of morphine, $\frac{1}{6}$ gr., given strictly four-hourly to cases of incipient ileus—that is, cases in which distension and vomiting are present. They also illustrate the fact that when morphine injections are given for four or five days the bowels often open spontaneously.

Example 36.—A woman aged 59 years had had four previous operations for obstruction and adhesions following an original operation of hysterectomy. When seen on this occasion she had been vomiting for a day, was considerably distended, and repeated enemas had produced no result. At operation numerous adhesions were divided throughout the lower small bowel. Following operation she had no audible bowel movements for two days, and after this she became somewhat distended. She was given $\frac{1}{6}$ gr. morphine four-hourly, and her bowels opened without aid on the sixth day after operation.

Example 37.—A man aged 35 years had an operation for acute appendicitis and generalizing peritonitis. A drainage tube was inserted. On the second day, when he became somewhat distended, $\frac{1}{6}$ gr. morphine was given four-hourly. His bowels were opened with a small enema on the fifth day after operation.

Example 38.—A man aged 59 years was operated upon for gangrenous appendicitis and general peritonitis. He became somewhat distended on the second post-operative day, when sounds could be heard in the abdomen. He was then given $\frac{1}{6}$ gr. morphine four-hourly. Following this the distension became less, he lost his feeling of acute abdominal discomfort, and his bowels were opened on the sixth day without the aid of enemas.

Example 39.—A woman aged 30 years had an operation for acute appendicitis with generalizing peritonitis. She was much distended before operation, and following operation continued to vomit and show marked distension, though she had tumultuous abdominal sounds on auscultation after the second day. She was given $\frac{1}{6}$ gr. morphine four-hourly. Her vomiting then ceased and her distension improved a little. Her bowels were opened six days later, after a small enema.

Example 40.—A woman aged 59 years complained that five hours before being seen she began to have colicky pains across the umbilical area, and soon after this to vomit. An indefinite mass could be felt above and to the left of the umbilicus. At operation a strangulated hernia into the paraduodenal fossa was reduced. It was only after considerable deliberation that it was decided not to resect. On the second day after operation she became distended; $\frac{1}{6}$ gr. morphine was given four-hourly, and from then her convalescence was satisfactory.

Treatment of Fully Developed Paralytic Ileus.—If distension is present and there are no movements to be found on auscultation, the patient has developed a paralytic ileus, and if he is not very distressed or upset, it is reasonable to treat him by giving morphine, $\frac{1}{6}$ gr. four-hourly, by passing an indwelling duodenal tube, and by attention to his fluid balance and serum protein concentration.

But in the case of a fully developed 'irreversible' paralytic ileus where the patient is very sick and distended, there is no question but that the use of the Miller-Abbott tube together with replacement therapy is the only way to save his life. It is also the only means by which an overdistended bowel can be reduced in calibre (that is, by the suction of a 'travelling ileostomy') to such a size that once more normal absorption from the loops can be established. It is in fact the only way of breaking the vicious circle of distension and lack of absorption in the stage of 'irreversible' ileus.

The adoption of the Miller-Abbott tube method often hinges on the difficulty of introducing the tube. The methods which will facilitate its introduction have been discussed in detail in a paper by John Devine (1945). There are very few cases in which with patience and practice it cannot be introduced with reasonable certainty into the duodenum by one of the methods described. Since the Miller-Abbott tube is the only means of curing a fully developed irreversible paralytic ileus, its introduction should be certain. Yet all figures quoted show that in a large number of the cases the tube could not have passed through the pylorus. Thus Smith (1945) mentioned that, despite the use of mercury in the balloon, for 1000 cases where intubation was attempted there were 221 failures. Other authors also publish a high percentage of failures.

In the present series of 43 successful intubations 41 were successful by the oral route. In this series were two cases in which the patients had a paralytic ileus, and were desperately ill. One had had the tube successfully passed twice before, but he had become so ill that it could not be passed a third time orally. The other had three times pulled out a tube which had been successfully introduced through the nose. In both instances the tube was then introduced through a small jejunostomy opening made under local anæsthesia.

To introduce the Miller-Abbott tube through a jejunostomy opening, when there is difficulty in passing it through the pylorus, is a logical procedure. It has been pointed out that an ileostomy in these patients is not sound treatment; in one case paralytic ileus actually developed after this operation. It is logical, therefore, that if in a desperately ill patient a Miller-Abbott tube, which provides a 'travelling ileostomy', cannot be passed by the normal way, and is needed for the patient's recovery, it should be

introduced through a jejunostomy. Temporary jejunostomy has been practised as a routine following gastric operations by some surgeons who prefer it to subjecting their patients to the annoyance of a nasal Abbott-Rawson tube. The opening in the jejunum should, however, be made as high as possible so as to empty the whole jejunum. In both the cases cited below, the Miller-Abbott tube successfully emptied the small intestine and reduced the distension. But in the first case death occurred later from an operatively unrelieved mechanical intestinal obstruction; and in the second from lung embolus. There does not appear to be any mention in the literature of this route for the introduction of the Miller-Abbott tube, which in many cases may be preferred by both surgeon and patient to the usual oral method.

Records of Cases in which the Miller-Abbott Tube had to be Introduced through a Jejunostomy.—

Example 41.—There had been bleeding following a gynæcological operation, and packing had been introduced into the pelvis, resulting in a paralytic ileus which was relieved by intestinal intubation with a Miller-Abbott tube in the normal manner. Peritonitis developed four or five days later and a Miller-Abbott tube was again passed. After four days more this appeared to have been controlled by chemotherapy and the tube was removed. However, distension once more occurred, and on this occasion, as the patient was very ill, it was found impossible to get the tube to pass the pylorus, though by means of a stylet and X-ray control it was kept against the pylorus for twenty-four hours. The tube was then passed through a jejunostomy under local anæsthesia, and eight hours later the first peristaltic movements were noticed on the recording apparatus, and thenceforth the tube was introduced at the rate of 4 in. an hour. Gas and fluid were withdrawn and the distension relieved. Subsequently the patient was operated upon under a local anæsthetic and a mass of adherent intestines was found and an ileostomy performed. Pneumonia supervened and death occurred several days later.

Example 42.—The case of a boy aged 19 years, who had an ileostomy performed as a preliminary to colectomy. Paralytic ileus supervened on the fourth day. On the fourth night the Miller-Abbott tube was passed, and by next morning was apparently coiled in the stomach and not through the pylorus (*Fig.* 239). Further attempts were made to pass the tube through the pylorus, and by that evening it was apparently through. However, at this stage the patient pulled out the tube. It was again introduced, and again after six or seven hours, when it appeared to be through the pylorus, it was pulled out by the patient. On the sixth post-operative day, under local anæsthetic, the tube was introduced through a jejunostomy (*Fig.* 240). A small right upper paramedian incision was made, and a very dilated dusky loop of intestine presented. This was deflated with a hypodermic needle fitted to the sucker, and then this was sewn into the peritoneal opening with a continuous catgut suture. Two purse-strings were then put in the bowel, which was opened, and the Miller-Abbott tube introduced to a distance of 3–4 in. A long silk suture was then attached to a big bite of bowel and let out through the wound so that when the tube was being pushed in counter-traction could be made on the thread. The wound was sewn up in the usual way.

Four hours after the operation, peristalsis was observed on the recording mechanism (*Fig.* 238), and thereafter the balloon was introduced at the rate of 3 in. an hour, and 40 oz. of fluid of a dark-brown colour was removed, together with the passage of much flatus, and the distension of the lower abdomen disappeared (*Fig.* 237). However, the upper part of the abdomen remained distended, and when a nasal Rehfuss tube was passed, it removed 128 oz. of dark-brown stomach contents. Following removal of these contents, peristalsis in the small intestine became much more marked. The patient was, meanwhile, given intravenous serum and saline in an endeavour to catch up on the huge negative fluid balance.

After two days of intestinal decompression by the Miller-Abbott tube through the jejunostomy opening, the tube was withdrawn to the 75-cm. mark and the patient fed with 2 oz. of glucose egg-flip every two hours through the suction lumen. His condition appeared to improve, but large quantities of fluid were still being periodically sucked out of his stomach. Successive gastric aspirations removed 100, 60, and 28 oz.

On the ninth post-operative day, three days after the intestinal intubation through the jejunostomy, the patient once more became slightly distended, and when the balloon was blown up no peristalsis was observed. At this stage another attempt was made to pass a second Miller-Abbott tube through the stomach from the mouth. This had successfully passed the pylorus when the patient (always difficult) pulled out the tube once more. After a difficult twelve hours, during which no peristalsis could be recorded from the balloon on the Miller-Abbott tube, which had been introduced through the jejunostomy, peristalsis once more commenced, and again the Miller-Abbott tube was passed through the small intestine at the rate of 4 in. an hour, this time removing much more normal looking small-bowel contents. The ileostomy below now commenced to work, and after the small intestine had been emptied by the Miller-Abbott tube it was withdrawn to the 75-cm. mark and used as a jejunostomy feeding tube and the patient fed two-hourly with glucose egg-flips.

On the eleventh day the patient had a perfectly flat and soft abdomen, and jejunostomy and ileostomy were both acting freely, but on the twelfth day he died suddenly from lung embolus.

This case illustrates the fact that the jejunostomy method of introduction of the tube is useful because besides being a method of introduction of the tube in difficult cases, it forms a useful way of feeding the patient in a case with acute gastric dilatation.

It should be pointed out that the Miller-Abbott tube should not be used for all cases of incipient ileus, as there are difficulties and dangers in its use. Also it is unpleasant for the patient to have for a long time an indwelling tube of fairly thick calibre which gives rise to pharyngeal and laryngeal irritation and, if used intranasally, to marked rhinitis. However, in the 'irreversible' stages of ileus, its employment should not be withheld too long. Further, it is no more annoying to the patient than an ordinary stomach tube, and even if it is not made to pass out of the stomach to the small bowel, it at least does the work of a stomach tube and has the potentiality of doing more.

Replacement Therapy.—The patient with paralytic ileus is indeed a 'test-tube baby', for when large amounts of fluid are withdrawn suddenly from the bowel, sudden dehydration and a condition resembling shock may supervene. Though the intestinal fluid is theoretically outside the circulation, there appears to be a constant 'give and take' of fluid through the bowel wall, which appears to be less selective and more "permeable" as a result of distension.

The route of replacement is important. Since saline, glucose, and serum are usually required, the intravenous route is the one of choice.

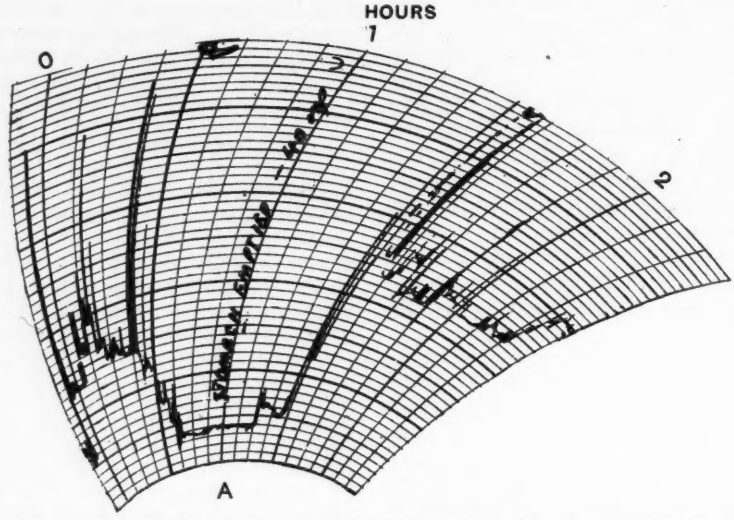


FIG. 237.—*Example* 42. To show the effect on peristalsis of distension of the stomach and its relief at point A.

Careful charting of the fluid balance is essential —it is too late when œdema or crepitations at the lung bases herald hyperhydration.

Glucose is probably absorbed in the colon in amounts insufficient to stimulate the pancreas, and if anything lowers the blood-sugar (Ebeling, 1933; Pressman, 1930), but the higher the concentration of saline the slower the absorption; therefore it is better to give tap water. In any case, if absorption is not immediate it may act as a bowel wash-out and stimulate small-bowel peristalsis.

Serum should be administered as the serum-protein is usually low. A very quick estimation of the serum-protein concentration can be made by the copper sulphate method, which has been used as a

FIG. 238.—*Example* 42. To show the first movements commencing five hours after laparotomy under local anæsthetic to place the Miller-Abbott tube in the jejunum.

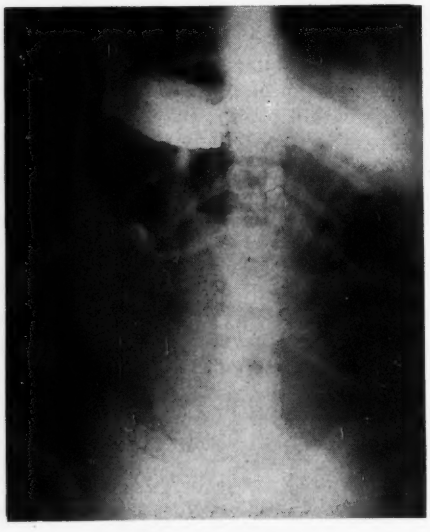


FIG. 239.—*Example* 42. Showing Miller-Abbott tube coiled in the stomach and small stomach pressed up under the diaphragm by distension.

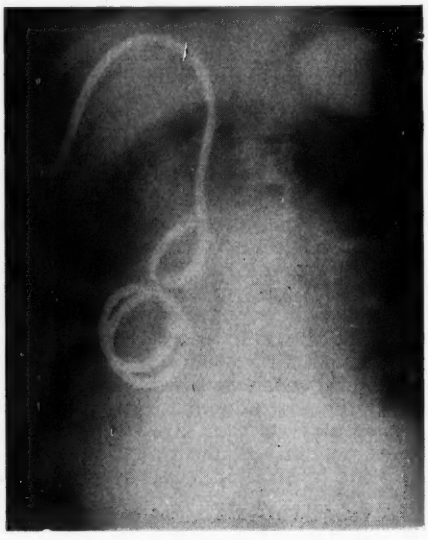
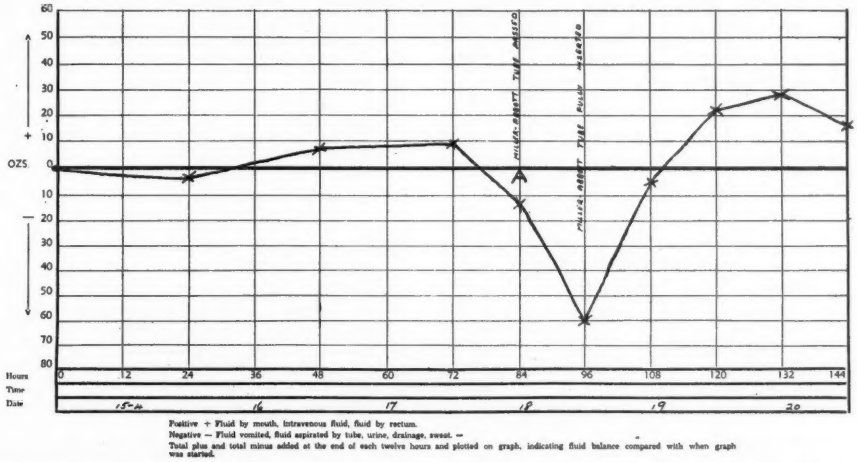


FIG. 240.—*Example* 42. The tube has been introduced into the loop of jejunum through the jejunostomy made in the abdomen under a local anæsthetic. The lower abdomen is being compressed, but several loops of upper jejunum can still be seen to have a ladder pattern present.

routine by the author in all cases where intestinal intubation has been performed (Van Slyke et al., 1944). This method requires at most 2 c.c. of blood and takes about a minute to centrifuge, after which a drop of the serum or plasma is dropped off a pipette into copper sulphate of various strengths. Copper sulphate and the protein in the serum form an insoluble compound, and for twenty or thirty seconds the drops either rise or fall, depending on whether they are in a solution of equal specific gravity or not. They then dissolve and the solution can be used again and again. Knowing the density of the solution in which the drop hangs suspended, the specific gravity of the serum or plasma is immediately apparent, and from it the serum-protein or plasma-protein concentration can be read off on a table. The patient is also put on a water balance chart (*Fig.* 241).



Positive + Fluid by mouth, intravenous fluid, fluid by rectum.
Negative — Fluid vomited, fluid aspirated by tube, urine, drainage, sweat. —
Total plus and total minus added at the end of each twelve hours and plotted on graph, indicating fluid balance compared with when graph was started.

FIG. 241.—To show how the fluid balance was recorded in a case of paralytic ileus—intravenous fluids were being administered continuously—a Miller-Abbott tube was passed at A.

Serum should be given at the rate of at least a litre a day, even though the serum-protein is not initially low, as very little serum is being absorbed from the bowel.

Wood and Bick (1941) found sodium chloride deficiency and some degree of acidosis in six of the cases they studied of paralytic ileus obstruction. Chew Smith and Van Beuren (1943) state that with the Miller-Abbott tube during the acute phases of ileus fluid replacement should average 2900 c.c. per day.

Quite apart from the value of serum and sodium chloride solutions in replacement therapy, they increase intestinal peristalsis.

The type of intravenous saline and glucose used should be considered. It has been found that the average salt content of the intestinal fluid removed in obstruction is 0·6 per cent (Abbott, 1942). Thus it is usual to give two litre flasks of 5 per cent glucose in saline followed by one of 5 per cent glucose, thus giving back to the patient a fluid with the same amount of saline content as the aspirated fluid, and thus restoring the fluid and saline balance.

When the Miller-Abbott tube has passed down the bowel from the stomach the patient may be fed above the tube, for glucose at least is absorbed in the duodenum and upper jejunum, but too high a glucose concentration is undesirable (Shay, Gershon-Cohn, Fels, and Munro, 1940).

The most striking evidence of the duodenal absorption of glucose occurred when, as a result of a congenital defect, a baby had only a few inches of duodenum brought out on to the abdominal wall and no remaining small bowel. Marked rise in the blood-sugar level occurred when glucose solution was fed into the duodenum. (Hendtlass, 1943.)

When the Miller-Abbott tube is down the intestine it is most desirable that the patient be given a high-carbohydrate and high-protein diet. A most useful method of accomplishing this has been the giving of glucose egg-flips made with citrated milk. This solution has also been used in two cases in which the Miller-Abbott tube was introduced through a jejunostomy.

When a tube is down, there is a marked loss of bile-salts, which may be replaced by capsules containing bile-salt given by mouth. Vitamins B and C are also indicated.

SUMMARY

1. The results of the clinical study of a number of cases of paralytic ileus, and of 43 cases in which intestinal intubation with the Miller-Abbott tube was performed, and of a number of observations on patients with ileostomies and jejunostomies and X-ray observations, are presented.

2. True or idiopathic post-operative paralytic ileus is rare. It is a dangerous diagnosis to make in a case of post-operative ileus; for it often serves as a solace to the pride, and prevents a correct diagnosis of the real cause till it is too late for appropriate treatment.

3. As a result of a study of drugs and procedures that might influence small-bowel motility in the prevention or treatment of paralytic ileus, it was found that :—

a. Heat or turpentine stupes on the abdomen had no effect on bowel movements.

b. Barbiturates (luminal) decreased peristalsis and tone.

c. Penicillin and sulphonamides did not materially affect peristaltic movements.

d. Over-distension of the stomach caused loss of tone and decreased movement of the small bowel.

e. Stimulation of the large intestine by enemas also stimulated the small intestine, even when this was completely isolated from the colon.

4. By far the most frequent cause of a post-operative ileus is a degree of post-operative intra-abdominal infection.

5. From a study of Miller-Abbott recordings and abdominal sounds in regard to the return of intestinal movements after operation, and the return of these movements during recovery from paralytic ileus, emanates what the author regards as an important hypothesis: viz., that there are three phases of ileus: Stage 1, in which the movements are active, co-ordinated, responsive to stimulation, and effective; Stage 2, when they become irregular, inco-ordinated, and are ineffective; and Stage 3, when the intestinal movements disappear and completely fail—in other words a fully-developed paralytic ileus. Stimulation in the second stage will often precipitate a fully-developed paralytic ileus.

6. A rational scheme of prophylactic and active treatment for paralytic ileus is based on these three stages.

7. Two cases in which the Miller-Abbott tube was introduced through a jejunostomy under local anæsthetic are reported. It is considered that in desperate cases jejunostomy and the introduction of a Miller-Abbott tube into the bowel by this route is more logical than ileostomy, which drains only the loop in which the opening is placed.

8. In view of the potential danger of such weapons as the enema and cathartics when given at the wrong stage, the delegation of the responsibility for their use to the nursing staff is much to be deplored.

I wish to thank Professor R. D. Wright, of Melbourne University Physiology School, for never-failing help and stimulative criticism, and the many nurses and sisters, particularly those of the " Mercy " Hospital, for their help in the arduous nursing and detailed recording.

APPENDIX

Adynamic Ileus.—

I. Intra-abdominal :—
- *A.* Peritoneal irritation :—
  1. Traumatic :—
     - *a.* Post-operative.
     - *b.* Penetrating wounds.
  2. Bacterial : Peritonitis.
  3. Chemical :—
     - *a.* Extravasation of blood.
     - *b.* Perforated peptic ulcer.
     - *c.* Bile peritonitis.
     - *d.* Acute pancreatitis.

     } EARLY.
- *B.* Vascular changes:—
  1. Strangulation :—
     - *a.* Intramural : Distension following mechanical ileus.
     - *b.* Extramural : Compression of the mesenteric vessels.
  2. Mesenteric thrombosis.
- *C.* Extraperitoneal irritation :—
  1. Hæmorrhage.
  2. Infection.
  3. Renal.

II. Extra-abdominal :—
- *A.* Toxic :—
  1. Pneumonia.
  2. Uræmia.
  3. Empyema.
  4. Systemic infection.
- *B.* Neurogenic :—
  1. Injuries and diseases of the spinal cord.
  2. Lead poisoning.
  3. Fracture of the lower ribs.

BIBLIOGRAPHY

AARAN, A. H. (1945), *J. Amer. med. Ass.*, **127**, 1027.
ABBOTT, W. O. (1941), *New Engl. J. Med.*, **225**, 641.
— — (1942), *Advances of Internal Medicine*, **1**. New York : Interscience Publishers Inc.
— — and JOHNSTON, C. G. (1938), *Surg. Gynec. Obstet.*, **66**, 691.
— — and MILLER, T. G. (1934), *Amer. J. med. Sci.*, **187**, 595.
— — and PENDERGRASS, E. P. (1936), *Amer. J. Roentgenol.*, **35**, 289.
ADLER, H. F. ATKINSON, A. J., and IVY, A. C. (1942), *Arch. intern. Med.*, **69**, 974.
ALLEN, K. D. A., and JAMISON, JOHN H. (1940), *Radiology*, **35**, 544.
ALVAREZ, W. C. (1918), *Surg. Gynec. Obstet.*, **26**, 651.
— — (1928), *The Mechanics of the Digestive Tract*. New York : Hoeber Inc.
— — and TAYLOR, F. B. (1917–8), *J. Pharmacol.*, **10**, 365.
ANTONCIC, R. F., and LAWSON, H. (1941), *Surg. Gynec. Obstet.*, **72**, 728.
ASHCROFT, P. B., and SAMUEL, E. (1940), *Lancet*, **2**, 445.
BARTELS, R. N. (1943), *J. Iowa med. Soc.*, **33**, 148.
BARDEN, R. P., THOMPSON, W. D., RAVDIN, I. S., and FRANK, I. L. (1938), *Surg. Gynec. Obstet.*, **66**, 819.
BAYLISS, W. M., and STARLING, E. H. (1899), *J. Physiol.*, **24**, 99.
BENNETT, L. C. (1941), *West. J. Surg.*, **49**, 71.
BOCKUS, H. L. (1944), *Gastroenterology*, **11**, 262.
BODENHEIMER, M., CASTEN, D., and FRIED, J. J. (1940), *J. intern. Coll. Surg.*, **3**, 28.
BOON, T. H. (1940), *Lancet*, **1**, 7.
BUIRGE, R. E. (1944), *Surgery*, **16**, 356.
BURGET, G. E., MARTZLOFF, K., SUCKOW, G., and THORNTON, R. C. B. (1930), *Arch. Surg.*, **21**, 829.
CARLSON, H. A. (1930), *Proc. Soc. exp. Biol., N.Y.*, **27**, 777.
CHEW SMITH, B. (1945), *Ann. Surg.*, **122**, 253.
— — and VAN BEUREN, F. T. (1943), *Ibid.*, **117**, 427.
CRANDALL, L. A., jun., CHESLEY, F. F., HANSEN, D., and DUNBAR, J. (1939), *Proc. Soc. exp. Biol., N.Y.*, **41**, 472.
CROHN, N., OLSON, W. H., and NECHELES, H. (1944), *Surg. Gynec. Obstet.*, **79**, 41.
DAVIS, H. H., and HANSEN, T. M. (1945), *Surgery*, **17**, 492.
DAVID, V. C., and LORING, M. (1930), *Ann. Surg.*, **92**, 721.
DEVINE, JOHN (1940), *Med. J. Aust.*, Sept. 7, 214.
— — (1945), *Ibid.*, 213.
DOUGLASS, D. M., and MANN, F. C. (1941), *Brit. med. J.*, **1**, 227.
DRAGSTEDT, C. A., LANG, V. F., and MILLETT, R. F. (1929), *Arch. Surg.*, **18**, 2257.
DUNCAN, J. A. (1940), *Northw. Med., Seattle*, **39**, 177.
EBELING, W. W. (1933), *Arch. Surg.*, **26**, 134.
FANTUS, B., KOPSTEIN, G., and SCHMIDT, H. R. (1940), *J. Amer. med. Ass.*, **114**, 404.
FELDBERG, W., and SOLANDT, O. M. (1942), *J. Physiol.*, **101**, 137.
FLOREY, H. W., WRIGHT, R. D., and JENNINGS, M. A. (1941), *Physiol. Rev.*, **21**, 36.

FORSTER, A. C. (1940), *Ann. Surg.*, **112**, 370.
GLEN, P. M. (1941), *Amer. J. Digest. Dis.*, **8**, 35.
GRIMSON, K. S., and HODGE, G. B. (1944), *Surg. Gynec. Obstet.*, **78**, 316.
GRUBER, C. M., GREENE, W. W., DRAYER, C. S., and CRAWFORD, W. M. (1930), *J. Pharmacol.*, **38**, 389.
— — and PIPKIN, G. (1930), *Ibid.*, **38**, 401.
HAAG, H. B., and TALIAFERRO, I. (1940), *Proc. Soc. exp. Biol., N.Y.*, **45**, 479.
HANEY, H. F., ROLEY, W. C., and COLE, P. A. (1939), *Amer. J. Physiol.*, **126**, 82.
HARTMAN, H., and DOCK, W. (1927), *J. Lab. clin. Med.*, **12**, 430.
HELM, J. D., jun., and INGELFINGER, F. J. (1944), *Surg. Gynec. Obstet.*, **79**, 553.
HENDTLASS, R. F. (1943), *Brit. med. J.*, April 17, 477.
HUGHSON, W., and SCARFF, J. E. (1924), *Bull. Johns. Hopk. Hosp.*, **35**, 197.
HUIDOBRO, F., MONTERO, E., and CUEVAS, F. (1944), *Surg. Gynec. Obstet.*, **78**, 471.
HUNT, C. J. (1944), *Radiology*, **43**, 107.
KANAN, M. A. (1937), *Proc. Soc. exp. Biol., N.Y.*, **36**, 506.
KAPLAN, I. W., and MICHEL, M. L. (1941), *New Orleans med. surg. J.*, **93**, 558.
KARABIN, J. E. (1942), *Amer. J. Surg.*, **56**, 471.
KENNING, J. C., and LOFSTROM, J. E. (1937), *Amer. J. Roentgenol.*, **37**, 28.
KEYES, E. L., jun. (1945), *Surgery*, **17**, 270.
KIEFER, E. D. (1944), *Gastroenterology*, **3**, 388.
KLEITSCH, W. P., and PUESTOW, C. B. (1939), *Surgery*, **6**, 679.
LADD, W. E., and GROSS, R. E. (1941), *Abdominal Surgery of Infancy and Childhood*, 195. Philadelphia: W. B. Saunders Co.
LAWSON, H. (1940), *Proc. Soc. exp. Biol., N.Y.*, **45**, 5.
LEIGH, O. C. (1942), *Surg. Gynec. Obstet.*, **75**, 279.
LEVITIN, J. (1942), *Medico-Surgical Tributes to Harold Brunn*, 253. Univ. Calif. Press.
LOFSTROM, J. E., and NOER, R. J. (1940), *Radiology*, 35, 546.
MCIVER, M. A., BENEDICT, E. B., and CLINE, J. W. (1926), *Arch. Surg.*, **13**, 588.
— — REDFIELD, A. C., and BENEDICT, E. (1926), *Amer. J. Physiol.*, **76**, 92.
MCLOUGHLIN, C. J., MANN, F. C., and KRUSEN, F. H. (1941), *Arch. phys. Ther.*, **22**, 325.
MARDEN, P. A., and WILLIAMSON, E. G. (1939), *Surg. Gynec. Obstet.*, **69**, 61.
MILLER, T. G. (1944), *Gastroenterology*, **3**, 141.
— — and KARR, W. G. (1936), *Amer. J. Roentgenol.*, **35**, 300.
MOIR, P. J. (1943), *Practitioner*, **151**, 71.
MYERS, B. N., and DAVIDSON, S. W. (1938), *J. Hyg., Camb.*, **38**, 432.
NOER, R., and JOHNSTON, C. G. (1942), *Ann. Surg.*, **115**, 935.
NOVIKOV, G. M. (1939), *Vestn. khir.*, **58**, 506; abstracted by *Int. Abst. Surg.*, 1940, **71**, 131.
OPPENHEIMER, M. J., and MANN, F. C. (1941), *Amer. J. digest Dis.*, **8**, 90.
OCHSNER, A. (1937), *Amer. J. Roentgenol.*, **37**, 433.
— — GAGE, I. M., and CUTTING, R. A. (1930), *Arch. Surg.*, **21**, 924.
— — — — — — (1933), *Ibid.*, **27**, 742.
— — JOHNSTON and HARVEY, J. (1945), *Surgery*, **17**, 873.
PENBERTHY, G. C., NOER, R. J., and BENSON, C. D. (1940), *Surg. Gynec. Obstet.*, **71**, 211.
PICK, E. P., BROOKS, G. W., and UNNA, K. (1944), *J. Pharmacol.*, **81**, 133.
PLANT, O. H., and MILLER, G. H. (1926), *Ibid.*, **27**, 361.
PRESSMAN, J. J. (1930), *Amer. J. med. Sci.*, **179**, 520.
PUESTOW, C. B. (1942), *J. Amer. med. Ass.*, **120**, 903.
QUIGLEY, J. P., HIGHSTONE, W. H., and IVY, A. C. (1934, a), *J. Pharmacol.*, **51**, 308.
— — — — — — (1934, b), *Amer. J. Physiol.*, **108**, 151.
RAVDIN, I. S., and ABBOTT, W. O. (1940), *Int. Clin.*, **1**, 178.
— — STENGEL, A., and PRUSCHANKIN, M. (1940), *J. Amer. med. Ass.*, **114**, 107.
ROWNTREE, L. G. (1922), *Physiol. Rev.*, **2**, 116.
SCHNEDORF, J. C., and ORR, T. G. (1941), *Amer. J. digest. Dis.*, **8**, 303.
SHAW, F. H. (1942), *Aust. J. exp. Biol. Med.*, **20**, 117.
SHAY, H., GERSHON-COHN, J., FELS, S. S., and MUNRO, F. L. (1940), *Amer. J. digest. Dis.*, **7**, 456.
SLAUGHTER, D., and GROSS, E. G. (1938), *J. Pharmacol.*, **63**, 289.
SMITH, B. C. (1945), *Ann. Surg.*, **122**, 253.
STEELE, J. M. (1942), *Advance in Internal Medicine.* New York: Interscience Publications Inc.
STONE, H. B. (1937), *Surg. Gynec. Obstet.*, **65**, 383.
STOUT, G. (1933), *Amer. J. Surg.*, **19**, 283.
— — (1943), *Nav. med. Bull., Wash.*, **41**, 56.
STREICHER, M. H., and QUIRK, L. (1943), *Amer. J. digest. Dis.*, **10**, 179.
SVIER, H. J., and MANN, F. C. (1943), *Surgery*, **13**, 67.
THOMAS, J. E., and KUNTZ, A. (1926), *Amer. J. Physiol.*, **76**, 606.
VAN LIERE, E. J., NORTHRUP, D. W., and STICKNEY, J. C. (1944), *Amer. J. Physiol.*, **142**, 615.
VAN SLYKE et al. (1944), *Bull. Hyg. Lond.*, **19**, 140 (abstract).
WAGNER, G. A. (1919), *Berl. klin. Wschr.*, 1221.
WAKIM, K. G., and MANN, F. C. (1943), *Gastroenterology*, **1**, 513.
WANGENSTEEN, O. H. (1942, a), *Intestinal Obstruction*, 437. Springfield, Ill.: C. C. Thomas.
— — (1942, b), *Ibid.*, 2nd ed., 435.
— — (1942, c), *Ibid.*, 8.
— — REA, C. E., SMITH, B. A., and SCHWYZER, H. C. (1939), *Surg. Gynec. Obstet.*, **68**, 851.
WILLIAMS, B. W. (1927), *Lancet*, **1**, 907.
WISE, R. A. (1938), *Amer. J. Surg.*, **41**, 412.
WOOD, I. J., and BICK, M. (1941), *Med. J. Aust.*, **1**, 140.
WRIGHT, R. D., 1946, personal communication.
YOUMANS, W. B. (1944), *Gastroenterology*, **3**, 114.
— — AUMANN, K. W., and HANEY, H. F. (1939), *Amer. J. Physiol.*, **126**, 237.

RECONSTRUCTION OF BICEPS BRACHII BY PECTORAL MUSCLE TRANSPLANTATION

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THE problem of reconstituting the biceps brachii arises most often in dealing with the aftermath of anterior poliomyelitis and only occasionally as a result of trauma. More or less complicated procedures have been described, but the operation which is the subject of this paper is technically simple to perform and, in the one case in which it has been tried, the result has proved satisfactory enough to suggest that it might, with advantage, be more widely employed.

The pectoralis major has been called by Keith 'the climbing muscle' and it was conjectured that such an action could still be used after the origin of the muscle had been transferred to the biceps tendon in the arm providing the nerve-supply of the transplanted fibres could be kept intact. In mammals, Keith (1933) describes a third pectoral muscle—the pectoralis externus—arising from the fourth, fifth, and sixth ribs and costal cartilages beneath the axillary border of the pectoralis major—and states that in man this muscle is commonly fused with, and forms part of, the pectoralis major. On the basis of this statement it seems reasonable to expect the outer portion of the pectoralis major to have a nerve-supply separate from the remainder and that it would be possible to construct a viable transplant from this part of the muscle. These arguments appear to be justified by the findings at operation and by the clinical result of the operative procedure founded upon them.

CASE REPORT

On Oct. 14, 1944, a German soldier sustained a shell wound of his left upper arm which caused extensive laceration of the biceps and coracobrachialis muscles with musculospiral nerve palsy. By the time the patient had reached a casualty clearing station gas gangrene had developed in the damaged muscles. On Oct. 15 the wound was excised and the biceps and coracobrachialis muscles were completely removed; the torn ends of the musculospiral nerve were seen. The wound was left open. 180,000 units of anti-gas gangrene serum were administered and a course of penicillin given intramuscularly for four days. One month later secondary suture of the wound was performed and satisfactory healing was obtained. A full range of passive movement was maintained in the elbow, wrist, and finger-joints; the musculospiral palsy was complete in the forearm and the action of the triceps was weak.

Five and a half months after wounding tendon transplantation was performed to restore extension of the wrist and digits. The result was satisfactory, but supination was weak and pronation absent. At the elbow there was no active flexion, but by a combination of weak triceps action and gravity full extension was present.

The use restored to the hand was such that the patient was anxious that something should be done for his arm. Reconstruction of the biceps was therefore undertaken by the method to be described.

OPERATIVE TECHNIQUE.—The patient was placed in a supine position with the affected arm half abducted and supported in full extension on a side table. The first incision reached from the level of the apex of the axilla distally along the axillary border of the pectoralis major to the seventh rib. The skin and superficial fascia were reflected to expose the underlying pectoral muscle and to define its lateral border and its lower costal attachment. The origin of the exposed muscle from the anterior surface of the sixth rib, together with its covering fascia, was detached to provide a piece of tissue 2½ in. wide. By separating the pectoral fibres along a line parallel to the muscle's axillary border a transplant large enough to act as a substitute for the biceps was isolated. One of the main terminal branches of the lateral anterior

FIG. 242.—Showing the pectoral muscle turned upwards, exposing the lateral anterior thoracic nerve.

thoracic nerve, which first appears at the level of the intercostal space between the third and fourth ribs, runs distally along the deep aspect of the lateral part of the great pectoral muscle and supplies it; its calibre is about equal to that of an intercostal nerve. As the separated part of the muscle was turned upwards the loose fascia under it was stretched and the nerve assumed an approximately horizontal position and was easily seen and preserved (*Fig.* 242.).

A second incision was then made on the anterior aspect of the lower third of the upper arm and the remains of the biceps tendon were exposed and mobilized. With the aid of a pair of long-bladed forceps thrust up from the incision in the arm a subcutaneous tunnel communicating with the upper end of the first incision was fashioned, through which the pectoral transplant was threaded. The lower part of the chest incision was then sutured, leaving sufficient of the upper part open to allow the tension on the nerve supplying the transplanted

muscle to be observed during the remainder of the operation. It was found that the transplant could not be drawn down the arm to its fullest extent because its nerve became stretched over the floor of the axilla even when the arm was fully adducted. The elbow was then flexed to a right angle and the tendon stump was looped through the distal end of the transplanted muscle and sutured to itself. Additional sutures were used to close the breach in the muscle and to fix it securely to the

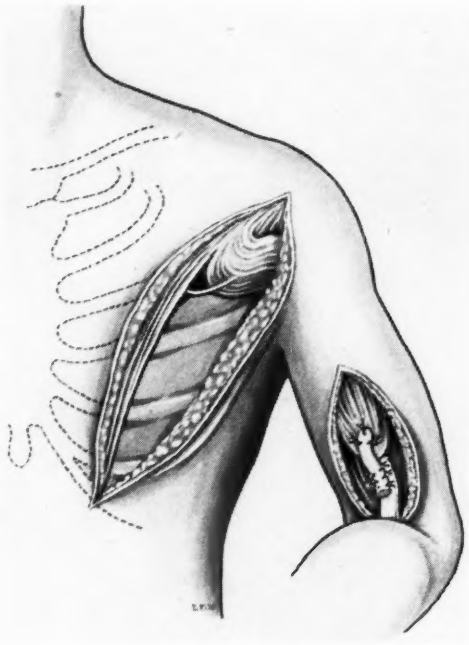

FIG. 243.—The muscle transplant completed.

penetrating tendon. (*Fig.* 243.) As shown in the diagram, the direct line of pull of the transplant was a little interrupted by the medial bulge caused by the stretched nerve, but there was no portion of the muscle sufficiently slack to suggest that its power would be vitiated. The incisions were closed and the arm was placed in a sling with the elbow maintained at a right angle.

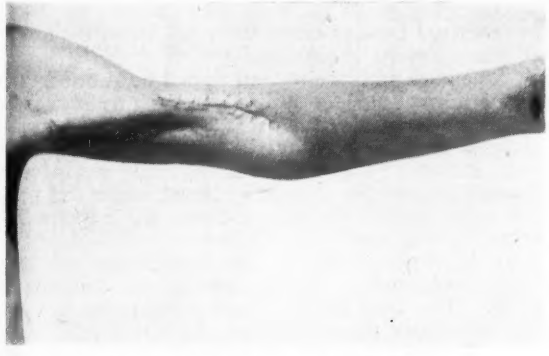

FIG. 244.—Showing the arm fully extended.

POST-OPERATIVE COURSE.—As soon as the patient was allowed out of bed he noted that, when walking and swinging his uninjured arm, he felt a little tug on the opposite forearm in the alternating phase of the swing. This demonstrated the integrity of the nerve-supply of the transplant, and kindled the patient's enthusiasm past damping. Immobilization in the sling was continued for three weeks and then, after removal of the skin sutures, active movements within the sling were started. The wounds were healed.

Six weeks after the operation full extension at the elbow was short only by 20° and active flexion to 70° was attainable; the transplant was palpably increased in size. Two weeks later the patient succeeded in flexing his elbow actively against a 7½-lb. resistance from its position of greatest extension to 20° short of a right angle—an excursion of 50°. The substitute muscle was now about three times its former size and on contraction closely simulated the action and appearance of the normal biceps. The arm was being used daily in the performance of sundry domestic duties.

Sixteen weeks after the operation the patient was repatriated, but before leaving he was examined by Major A. S. Bullough, R.A.M.C., who reported that full active flexion of the elbow was short only by 15° and that full extension was short by 5°, and he estimated that the power of flexion was 40 per cent of normal. Flexion of the elbow was accompanied by a shrugging of the shoulder, but the patient had grown accustomed to this action and could easily initiate the movement required. Photographs of the arm in positions held actively were also taken (*Figs.* 244, 245).

SUMMARY

1. A portion of pectoralis major was used as a transplant to replace the biceps brachii which had been destroyed.

2. The operative technique is described.

3. The case is reported fully.

REFERENCE

KEITH, SIR A. (1933), *Human Embryology and Morphology*.

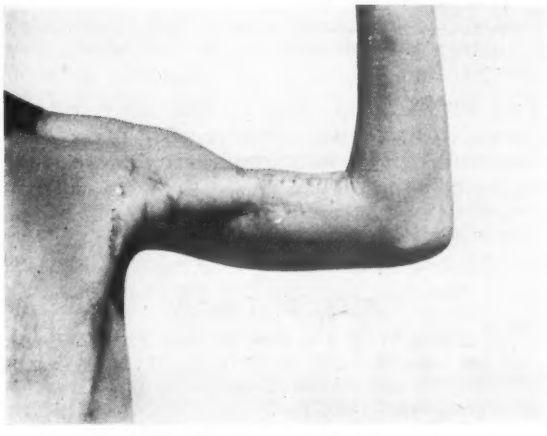

FIG. 245.—Showing the arm flexed to a right angle.

TRIPLICATE URETER

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THE incidence of duplication of the ureter (double and bifid ureters) is sufficiently common to lead any urologic clinic to expect at least a few cases each year, and has been reported in 0·5 per cent of autopsies (136 out of 26,480) by Meredith Cambell (1937) in his exhaustive book. Triplication (triple and trifid ureters) is, however, much rarer; Cambell found only 3 cases, those referred to and substantiated by Lau and Henline (1931), who also collected references to inadequate records of 3 other cases.

One new case of trifid ureter is therefore described here, and 10 previously recorded cases of triple and trifid ureter, together with 3 cases which showed more than three ureters on one side, have been collected, summarized, and briefly discussed.

FIG. 246.—Cystogram with patient upside down, showing two kinked hydro-ureters and two renal pelves.

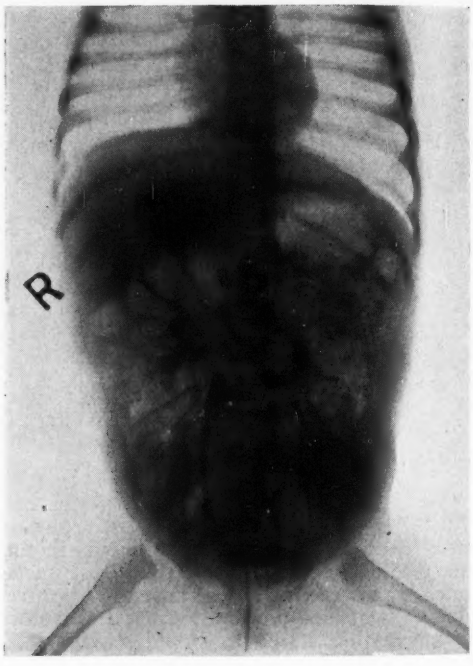

FIG. 247.—Retrograde pyelogram showing normal ureter and pelvis on the right.

CASE REPORT

C. P., a female aged 6 months, was brought to the Hospital for Sick Children on May 9, 1945, complaining of (1) frequent clay-coloured motions, (2) apparently painful defæcation. She had weighed 8 lb. at normal delivery at full term, had been breast-fed for two weeks, and since then had been on Cow & Gate (F.C.).

Examination showed a moderately ill baby weighing 13 lb. 5 oz.; scratch marks on scalp; abdomen tense and swollen with palpable hard masses of fæces; anus extremely tight; rectum very full; the urine contained albumin, pus, and *B. coli*. The mouth, fauces, tonsils, ears, chest, and heart were clinically healthy. The urinary infection was considered to be secondary to the alimentary stasis.

Intermittent dilatation of the anus, aperients, and rectal wash-outs soon restored the bowel to normal. A later attack of gastro-enteritis cleared up after a fortnight, but the urine was not sterilized by two consecutive courses of sulphathiazole with mist. pot. cit. alk., or by a subsequent course of mandelates with sod. acid. phosph.

On June 20 the urine still contained 70 mg. albumin per 100 c.c., and grew *Ps. pyocyaneus* as well as *B. coli*, and the blood-urea was 37 mg. per 100 c.c., so the patient was transferred back to Great Ormond Street for further investigation.

A cystogram with the patient tipped head downwards showed a normal bladder outline with two widely dilated and kinked ureters on the left side (*Fig.* 246). *Cystoscopy* on July 6 showed considerable cystitis, but only two ureteric orifices. The left orifice gaped considerably. *Retrograde pyelography* showed a normal ureter and pelvis on the right, though the pelvi-ureteric angle was a little accentuated (*Fig.* 247).

Left Nephro-ureterectomy was performed through a left loin incision by Mr. T. Twistington Higgins on July 18. The two dilated upper ureters were found arising one above the other from the upper pole of the kidney. A third ureter of normal dimensions and appearance was found arising from the lower pole (*Figs.* 248, 249). The appearance of the kidney suggested the possibility of two closely apposed kidneys. Subsequent dissection failed to prove this, but showed that the upper pole containing the upper two pelves consisted of whitish and scarred tissue without any characteristic naked-eye renal pattern, while the lower two-thirds were pinker, with normal cortical and medullary patterns. The three pelves arose separately one above the other in

the middle plane of the kidney (*Fig.* 250). At operation the ureters were dissected free as low as possible and divided at about 1 in. above the bladder. Down to this point the two thick ureters were enclosed in a common fibrous sheath, while the single healthy ureter remained quite separate from and lateral to the other two. How they entered the bladder could not be seen from above, but cystoscopy had showed only one ureteric orifice on each side, so it is assumed that this was a trifid ureter,

FIG. 248.—Kidney, as removed, showing the ends of two thickened dilated ureters and a third normal ureter.

FIG. 249.—Kidney showing two hydro-ureters from upper pole enclosed in a common fibrous sheath.

with the components uniting just above or actually in the bladder wall (*Fig.* 252, K).

Convalescence was complicated only by recurrence of slight fæcal retention and the baby was discharged home on July 31, aged 8½ months, with the urine sterile and free from albumin, and the general condition very good.

TYPES OF TRIPLICATE URETER

If three ureters arise from a kidney: (1) they may open into the bladder (or urethra or elsewhere) independently through three orifices, viz., complete

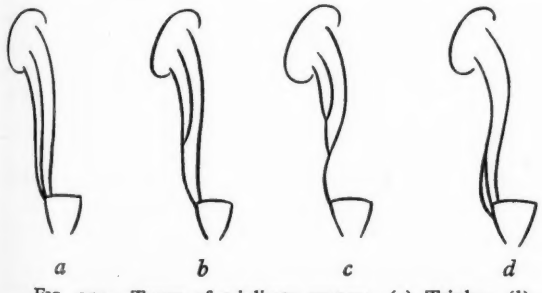

FIG. 251.—Types of triplicate ureter: (*a*) Triple; (*b*) Double with one bifid; (*c*) Trifid; (*d*) Double, with 'inversion Y' bifurcation.

triple ureter (*Fig.* 251, *a*); two ureters may unite, leaving only two orifices, a condition I have called 'Double ureter with one bifid' (*Fig.* 251, *b*)—I can find no record of any case showing a double ureter with both bifid; (3) all three ureters may unite and drain through a single orifice, viz., 'trifid ureter' (*Fig.* 251, *c*). If two ureters arise from a kidney, the bifurcation of one of them before it reaches the bladder would constitute a triple ureter; such inverse Y bifurcation has been reported four times in association with reduplicated ureters (Cambell, 1937), and it is possible that Chwalla's case (*see below*) is of this type (*Figs.* 251, *d*, 252, C).

FIG. 250.—Kidney, split in approximately the median plane, with a matchstick inserted through each pelvis into each ureter.

PREVIOUSLY RECORDED CASES

The following are summaries and diagrams of previously recorded cases arranged according to this scheme:—

Triple Ureter has been recorded four times, incidentally right-sided in each case. Only *Case* 1 was satisfactorily demonstrated as complete, in *Cases* 2 and 3 the origin of one ureter remained obscure; in *Case* 4 there was a vulval ectopic ureteric orifice.

Case 1 (Woodruff, 1941).—A man, aged 74, with right-sided triple ureter (*Fig.* 252, A) which was found accidentally with routine investigations prior to a routine prostatectomy. No symptoms. A complete case, the original article contains an excellent pyelogram showing all three pelves and ureters.

Case 2 (Lau and Henline, 1931).—A negress, aged 52, with right-sided triple ureter had complained of frequency, dysuria, with lumbar and crural pain for four years. *Cystoscopy* showed three ureteric orifices on the right. The first and second secreted dye in 10 min.; the third none, and could not be catheterized beyond 1 cm. *Retrograde pyelography* showed the first two draining a double kidney, and the third ending in an irregular small cavity in front of the lumbosacral junction. This was taken to represent an ectopic mal-developed accessory right kidney (*Fig.* 252, B).

Case 3 (Chwalla, 1935).—A man, aged 41, with a right-sided triple ureter was a tabetic being investigated for vesical calculi. *Cystoscopy* showed three ureteric orifices on the right, all secreting dye in 3–4 min. *Intravenous pyelography* showed on the right three ureters,

but only two renal pelves. *Retrograde pyelography* suggested that the two lateral ureters both drained the lower pelvis, and possibly represented the bifurcation of a single ureter at the level of L.4–5 disc (*Fig.* 252, C). The patient refused further investigation.

Case 5 (Wrany, 1870).—A girl, aged 3, with left-sided double ureter, one bifid, died of encephalitis and chronic subdural hæmatoma. *Autopsy* showed the left kidney rotated hilum forwards, with two pelves arising side by side in the lower half of the kidney, each giving rise to a

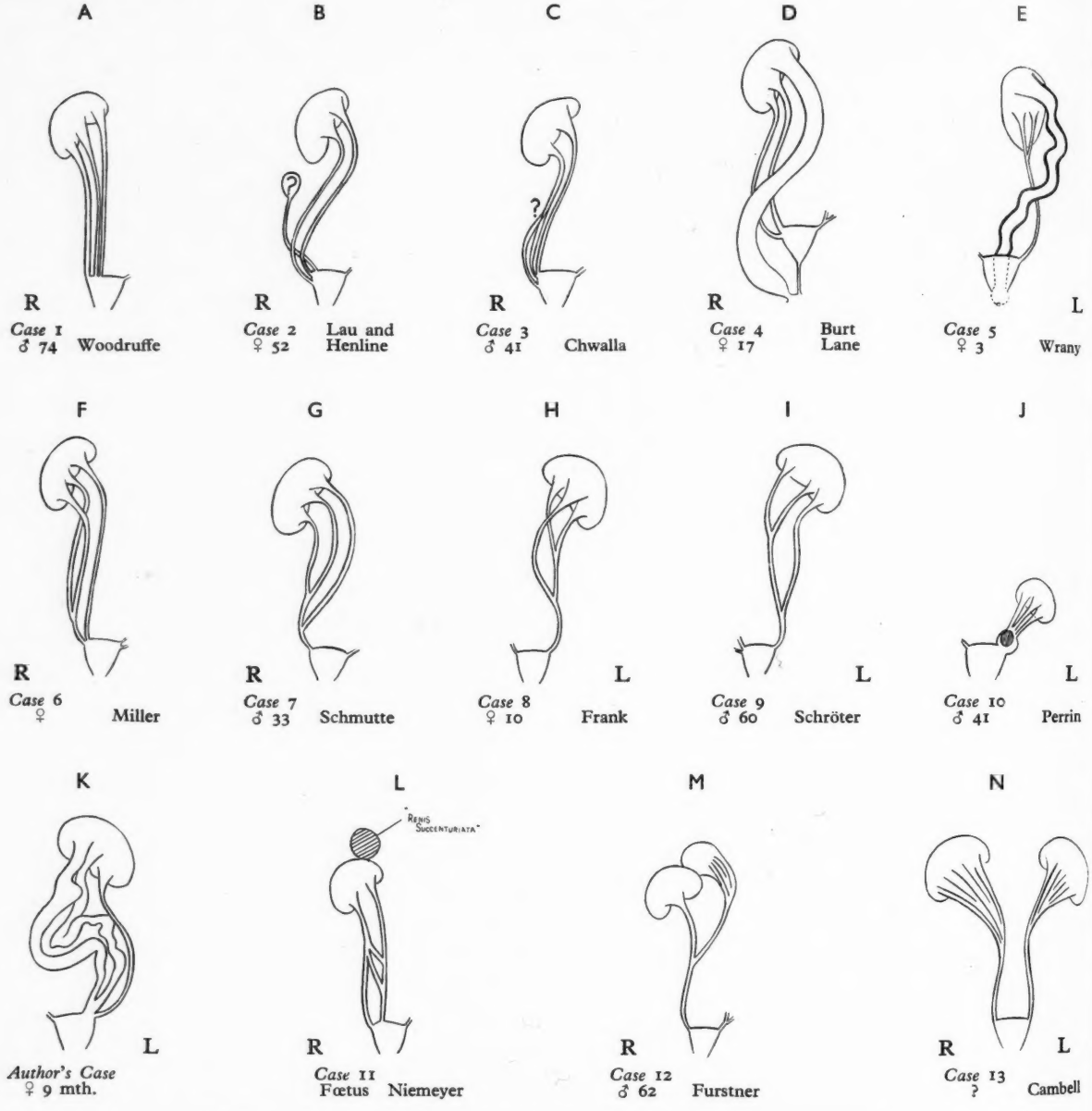

FIG. 252.—Diagrams showing the arrangement of the ureters in the cases reported.

Case 4 (Burt et al., 1941).—A girl, aged 17, with right-sided triple ureter was being investigated for incontinence since birth. *Cystoscopy* showed two ureteric orifices on the right, but intravenous injection of dye revealed a pin-point orifice in the vulva just to the right of the urethral meatus. *Intravenous pyelography* showed three pelves and three ureters on the right, the upper one being very dilated and leading to the ectopic orifice (*Fig.* 252, D). *Operation* showed three ureters arising from the pelves of a normal kidney.

Double Ureter with One Bifid has been recorded twice; in each case the lower ureter was bifid and the upper was separate.

normal ureter. These united 4 in. below the kidney. A third pelvis situated in the lower pole gave origin to a thick-walled tortuous hydro-ureter, which ran downwards lateral to the other two, crossed anterior to them, and opened into a membranous diverticulum of the bladder just above the urethra (*Fig.* 252, E). This diverticulum, hydro-ureter, and upper pelvis were heavily infected and the corresponding renal tissue was hydronephrotic.

Case 6 (Miller, 1938).—Miss H., with right-sided double ureter, with one bifid, complained of backache, and her urine grew *B. coli*. *Cystoscopy* showed two ureteric orifices on each side. *Retrograde pyelography*

showed three pelves and three ureters on the right; the two lower ureters, after crossing high up, united in front of the sacro-iliac joint (*Fig.* 252, F).

Trifid Ureter has been recorded four times. The pattern of trifurcation was different in each case.

Case 7 (Schmutte, 1929).—A man, aged 33, with right-sided trifid ureter, underwent an operation for subcapsular hæmatoma. The specimen removed was a small ectopic kidney, 7·5 × 3·2 cm., floating in a subcapsular hæmatoma low down on the anterior surface of the psoas. One pelvis in the upper pole and two pelves side by side in the lower pole each gave origin to a normal ureter. *Cystoscopy* later revealed only one ureteric orifice on the right (*Fig.* 252, G).

Case 8 (Frank, 1933).—A girl, aged 10, with left-sided bifid ureter and a patent interventricular septum, died of pneumonia after an operation for repair of epispadias. *Autopsy* showed three pelves arising one above the other from a normal sized left kidney. Each gave rise to a normal ureter, of which the upper and the lower united ½ in. below the kidney, and were joined 2 in. farther down by the third, thus entering the bladder on the left through a normal single orifice (*Fig.* 252, H).

Case 9 (Schröter, 1937).—The cadaver of a man, aged 60, with left-sided trifid ureter was dissected at Leipzig University. Each kidney has two "hiluses". The left upper hilus gave origin to a normal pelvis and ureter. The left lower hilus gave origin to two pelves and two ureters, one above the other and all appearing normal, except that the middle pelvis was much smaller than the other two. The two upper ureters united 16 cm. below the kidney and were joined 6 cm. above the bladder by the third (*Fig.* 252, I).

Case 10 (Perrin, 1927).—A man, aged 41, with left-sided trifid ureter and an ectopic left kidney had three attacks of left lumbar pain with frequency and hæmaturia. The urine grew streptococci. *Cystoscopy* showed blood coming from a single orifice on the left. *Pyelography* showed a sacral ectopic hydronephrotic small left kidney. Operation showed this lying in the hollow of the sacrum and giving rise to three ureters which soon united to form a small sac which drained into the bladder through one orifice and contained a large calculus. The outlets of the two upper ureters were obstructed by this stone. This specimen was demonstrated to the Royal Society of Medicine by Mr. Perrin on March 24, 1927 (*Fig.* 252, J).

Multi-branched Ureters have been reported, but the branching is usually very close to the kidney, and the ducts possibly extenuated calices of metanephric origin rather than true ureters.

Case 11 (Neimeyer, 1814).—A full-term female fœtus with four branches to its right ureter amongst many other congenital deformities was the thirteenth infant of a woman of 40. Neimeyer's diagram (reproduced in a simplified form in *Fig.* 252, L) does not tally closely with his text, which, literally translated, reads "... from the anterior side of the right kidney, which resembles a renal pelvis, proceeded four tubules separating at some points and uniting at others and passing beneath the rudimentary vagina . . ."

Case 12 (Furstner, 1897).—A man, aged 62, with a five-branched ureter died from pulmonary and miliary tuberculosis. *Autopsy* showed bilateral bifid ureters. The upper right ureter arose " . . . from five wide canals . . ." which began in the five calices of the smaller partly separate " appendage of the right kidney " (*Fig.* 252, M).

Case 13 (Cambell, 1937).—Bilateral six-branched ureters were found in an adult at autopsy at the Bellevue Hospital, New York. No further details were given (*Fig.* 252, N).

DISCUSSION

The clinical significance of these anomalies seems not much different from that of double ureters. In 5 cases (38 per cent) the condition was present without clinical manifestation. But there is an increased risk of obstructive hydro-ureter (3 cases) possibly leading to infection (3 cases), and pyonephrosis (2 cases). However, even if two badly infected ureters are found clinically, there remains the unexpected possibility, however remote, that a third and healthy may be found draining healthy renal tissue on the same side, as occurred in this new case. There is also an increased predisposition to stone formation (2 cases), and the tendency to association with other congenital abnormalities (3 cases), such as ectopic renal tissue (1 case), aberrant renal vessels (1 case), ectopic ureteric orifice (1 case), and these may all cause trouble.

The embryological significance I cannot fully assess. The theory that separate ureteric buds can arise independently from the primitive Woolfian duct seems to be supported by the existence of three ureteric orifices on one side of the bladder. The theory that complete double ureters may be formed simply by fission, however early, of one ureteric bud seems to be supported by finding: (1) two ureters enclosed throughout their course in a common fibrous sheath (the third being separate), (2) two renal pelves lying side by side with each other as well as one above the other in the same kidney. This supports the present general opinion that either or both of these embryological faults may take part in the formation of ureteral anomalies.

It is a pleasure to record my thanks to Mr. T. Twistington Higgins, O.B.E., for permission and encouragement to publish this case; to the librarians of the Royal Society of Medicine and the British Medical Association for help with references; and to Mr. Allevoyne, of the Photographic Department, Hospital for Sick Children, for his photographs.

REFERENCES

BURT, J. C., LANE, and HAMILTON, (1941), *J. Urol.*, **46**, 235.
CAMBELL, MEREDITH F. (1937), *Pediatric Urology*, I. New York.
CHWALLA (1935), *Z. urol. Chir.*, **41**, 224.
FRANK, A. (1933), *J. Urol. med. chir.*, **35**, 255.
FURSTNER, C. (1874), *Virchows Arch.*, **59**, 401.
LAU, F. T., and HENLINE, R. B. (1931), *J. Amer. med. Ass.*, **96**, 587.
MILLER, J. (1938), *Brit. J. Urol.*, **10**, 249.
NEIMEYER, C. E. (1814), " Singularis in foetu puellari recens edito Abnormitatis exemplum descriptum et illustratum ", *Inaug. Diss., Halle.*
PERRIN, W. S. (1927), *Proc. R. Soc. Med.* (Sect. Urol.), **48**.
SCHMUTTE, H. (1929), *Z. urol. Chir.*, **28**, 284.
SCHRÖTER, H. (1937), *Z. ges. Anat.* 1; *Z. anat. Entw-Gesch.*, **107**, 18.
WOODRUFFE, S. R. (1941), *J. Urol.*, **46**, 376.
WRANY, A. (1870), *Ost. Jh. Pædiat.*, **1**, 105.

THE SURGICAL ANATOMY OF THE PAROTID GLAND

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THE low incidence of facial paralysis following operation for parotid tumour was emphasized by Hamilton Bailey (1941), who reported 6 cases, in 5 of which the nerve escaped completely. More recently in a personal communication he records a series of over 30 complete or partial parotidectomies with only 2 cases of nerve damage. To account for these successes he cites the operative appearance of the gland, which in nearly all cases showed a large superficial lobe and a smaller deep lobe connected by an isthmus. Above and below the latter the primary divisions of the facial nerve could be seen running forward in the plane between the lobes. It was possible to remove the superficial lobe without disturbing the nerve in any way, and, when necessary, the deep lobe could also be extirpated by retracting the cervicofacial and temporofacial divisions of the nerve and extracting the gland tissue between them. On account of these observations, Hamilton Bailey suggests a revision of the standard anatomical descriptions of the gland, which make no mention of the bi-lobed nature of the organ.

As far back as 1912 Gregoire showed that the parotid was divisible into superficial and deep lobes separated by the branches of the facial nerve. He stated that the two lobes were united above the nerve. Observing that there was no deep lobe in the three-months' fœtus, he suggested that upward extension of the gland was halted on reaching the base of the skull and that further growth resulted in an inward and then downward deflection of the gland, with consequent production of a deep lobe.

McWhorter (1917) confirmed the bi-lobed nature of the gland, but found the connecting isthmus was situated not superiorly, but centrally, being embraced above and below by the temporofacial and cervicofacial branches of the nerve. This is in keeping with Hamilton Bailey's observations at operation, but is incompatible with Gregoire's theory concerning the origin of the deep lobe. More recently, McCormack, Cauldwell, and Anson (1945) have confirmed the existence of a centrally placed isthmus with branches of the facial nerve above and below it, but their report concentrates mainly on the variety of patterns formed by the terminal branches of the facial nerve.

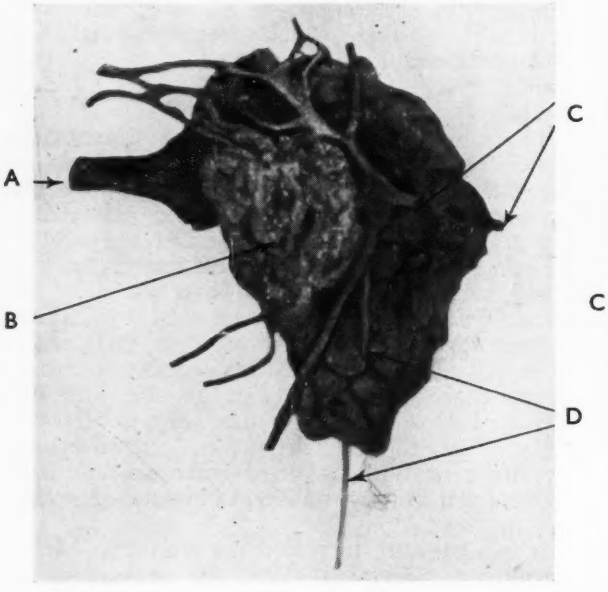

FIG. 253.—Medial aspect of parotid showing small deep lobe. Note that the trunk of the facial nerve is at first embedded in the substance of the superficial lobe, while the cervical branch also pierces the gland substance inferiorly. A, Parotid duct; B, Deep lobe; C, Trunk of nerve piercing superficial lobe; D, Cervical branch piercing superficial lobe.

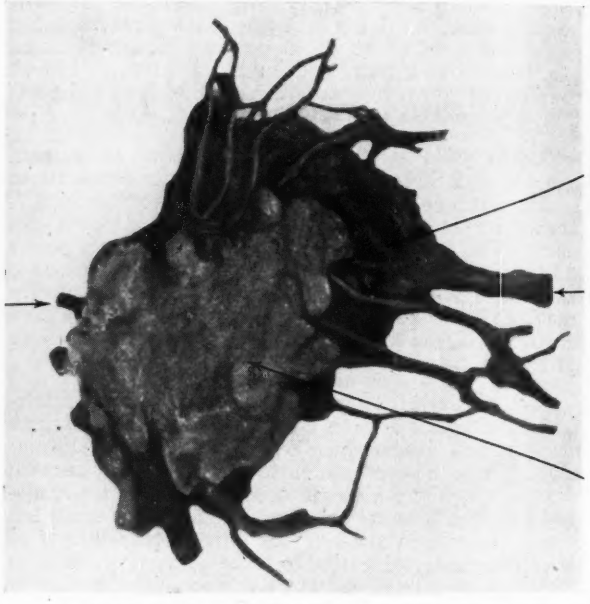

FIG. 254.—Lateral aspect of parotid, showing small superficial lobe and large deep lobe. A, Parotid duct; B, Deep lobe; C, Trunk of facial nerve; D, Superficial lobe.

The present series of eleven normal adult parotid glands were dissected to ascertain whether the bi-lobed nature of the organ was sufficiently well defined to permit of partial or total parotidectomy without nerve damage; in other words, to determine whether the normal anatomical lobes are comparable with those apparently discernible at operation on the diseased organ.

FINDINGS

In 10 of the 11 specimens there was evidence of division into superficial and deep lobes. In 7 of these the isthmus was clearly defined, being narrow in 6 and broad in 1; in the remaining 3 it was ill defined and its position inconstant. In all 10 specimens tedious and careful dissection was required to demonstrate the isthmus. Of the 10 bi-lobed parotids there were only 4 in which all the branches of the facial nerve remained strictly in the plane

between the lobes. In 5 one or more branches of the nerve pierced the deep lobe and in 1 some of the branches traversed part of the superficial lobe (*Fig.* 253). Moreover, in 3 specimens the trunk of the facial nerve before division lay not between the lobes, but in the substance of one or other lobe (*Fig.* 253). Finally, in 5 parotids the deep lobe was found to be larger than the superficial one (*Fig.* 254).

DISCUSSION

Contrary to Bailey's findings in the diseased parotid, the above observations show that it is often difficult to define the two lobes of the normal gland. Furthermore, when they have been identified, the branches of the facial nerve are not always confined to the interlobar plane, nor does the nerve-trunk invariably enter between the lobes. Definition of the two lobes would probably be easier in the living subject than in the formalin-preserved cadaver, but deviations of the nerve-trunk or some of its branches from the interlobar plane would still be encountered. In view of the highly successful operative results reported by Bailey, one is forced to the conclusion that the lobes encountered surgically are probably not strictly comparable with those of the normal gland. An encapsulated tumour growing in the superficial lobe would tend to thin out and compress the surrounding normal gland tissue and displace the nerve medially. Moreover, the size of the tumour compared with the uninvolved deep part of the gland might give the impression of a more well-marked isthmus than actually exists normally.

McWhorter (1917) and Bailey (1941) both refer to the smaller size of the deep lobe, and the latter author implies that superficial lobectomy alone adequately removes most neoplasms. However, in nearly half of the present series the deep lobe was the larger part of the gland and its removal without damaging the facial nerve would be a most difficult procedure and probably impossible in the presence of a tumour. In view of Bailey's successes it would appear, therefore, that the deep lobe is less liable to neoplastic change. There is, however, no obvious difference in the microscopical appearance of the normal lobes.

I wish to thank Professor T. Nicol for help and advice in preparing this paper and also Mr. Hamilton Bailey, whose personal communication to Professor Nicol led to the investigation.

REFERENCES

BAILEY, HAMILTON (1941), *Brit. J. Surg.*, **28**, 337.
— — (1945), personal communication.
GREGOIRE, R. (1912), *J. Anat., Paris*, **48**, 437.
MCCORMACK, L. J., CAULDWELL, E. W., and ANSON, S. J. (1945), *Surg. Gynec. Obstet.*, **80**, 620.
MCWHORTER, J. (1917), *Anat. Rec.*, **12**, 149.

SIMPLE PAPILLOMA OF THE URETER

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"PRIMARY tumours of the ureter are exceptionally rare" (Illingworth and Dick, 1941). Similar statements can be found in other text-books, and afford justification for recording even single cases. According to Moore (1942), whose review is the latest in the English literature, the first case of benign tumour of the ureter was published in 1861 by Lebert. The first account of a malignant neoplasm appears to be that of Rayer (1841), while Davy (1884) was the first to describe the condition in the English literature. There are less than 60 cases of benign tumour reported in the literature: Johnson and Smith (1942) brought the number up to 53, Moore (1942), and McMahon (1944), each reported 1 case, and Barnes and Kawaichi (1944) recorded 2 examples. Malignant tumours, while rare, are commoner than benign ones: Counseller, Cook, and Seefeld (1944) found 191 reported cases.

Benign tumours of the ureter occur on either side, and at any age, but are commoner in the fifth and sixth decades. They are commoner in men than in women, and appear in the lower portion of the ureter more often than in the upper. Usually they are papillomatous, closely resembling the much commoner papilloma of the urinary bladder: a few, such as those reported by Melico and Ford (1932) and Johnson and Smith (1942), are polypoid connective-tissue tumours. The papillary tumours may be single or multiple, are usually small, red or grey, soft, with a shiny surface; they arise from a distinct pedicle and are commonly villous. The ureter usually shows a fusiform dilatation at, and a little above, the level of the tumour. The presenting symptoms and signs in order of frequency are hæmaturia, pain, and renal enlargement. Diagnosis cannot be made without the aid of ureteric examinations, including intravenous and retrograde pyelography.

Nothing is known about the aetiology of these tumours, although Johnson and Smith (1942) thought that the polyps in their case were congenital, and Müller (1940) recorded a benign papilloma in a man of 42, who from the age of 18 to 26 handled benzidine in his work. Calculi are

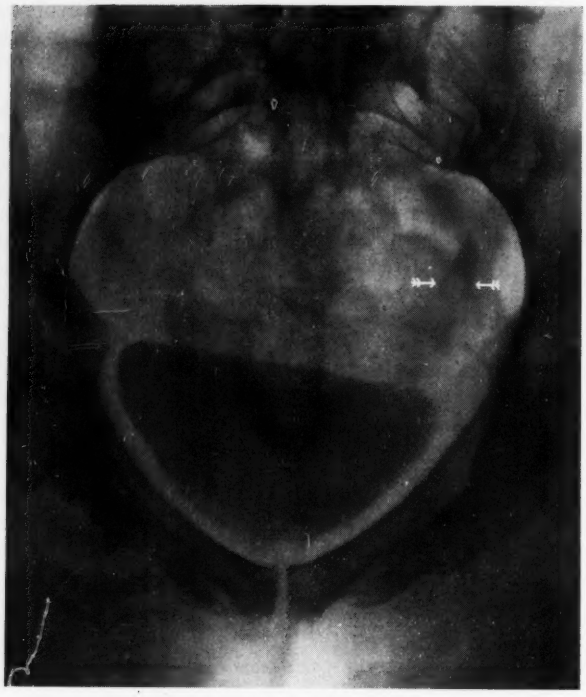

FIG. 255.—Intravenous pyelogram, positive print. Arrows indicate filling defect at lower end of left ureter.

FIG. 256.—Photograph of specimen. (*Reduced.*)

present in a little over 10 per cent of cases, and Melico and Ford (1942) considered they played a causative role in the production of the tumour in their patient: it is very doubtful, however, whether calculi are ever anything but incidental or secondary to the tumour.

FIG. 257.—Photograph of ureter and tumour. (*Natural size.*)

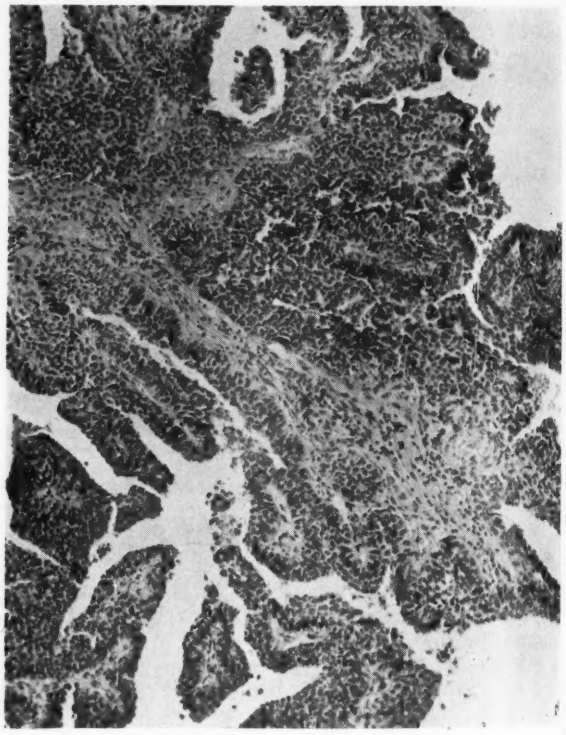

FIG. 258.—Photomicrograph of papilloma, stained H. and E. (× 90.)

CASE REPORT

Mrs. B., aged 42 years, was referred to one of us (H. B. Y.) by her doctor because of two recent painless attacks of hæmaturia. She had not experienced frequency, dysuria, or backache. Her blood-pressure was

normal, and no evidence was found of any constitutional disability likely to produce hæmaturia. There was neither renal tumour nor tenderness. On cystoscopy a little blood was seen coming with the urine from the left ureteric orifice: indigocarmine injected intravenously appeared from the right ureter in 4½ minutes, from the left it appeared in 6 minutes and the urine was purple instead of blue. A ureteric catheter was passed up the left ureter to the kidney pelvis, and, unexpectedly, no blood was found in the urine coming down the catheter. It was at first considered that there were two ureters on the left side and that the catheter had passed into the one not discharging blood.

A plain radiographic examination did not reveal any abnormality of the urinary tract nor any evidence of urinary calculus. Intravenous pyelography was undertaken and five minutes after the injection of the drug, normally functioning kidneys were seen, with no signs of hydronephrosis or other abnormality. The ureters were not clearly outlined. Firm pressure was then applied to the lower abdomen and further radiographs taken thirty minutes later revealed the ureters. The lower end of the left ureter ended abruptly in a sharp concave extremity at a point about 2 in. above the ureteric orifice. The shadow of the drug-laden urine was seen embracing a clear, semicircular area (*Fig.* 255). The appearances suggested either a papilloma or else a urate stone, which, in its pure state, is only slightly radio-opaque and cannot be demonstrated in a plain radiograph.

It was remembered that a slight arrest of the catheter had been felt about an inch up the ureter, and it was now evident that the bleeding must be coming from the lower end of the ureter and not from the kidney.

OPERATION was performed on June 28, 1945, under spinal anæsthesia plus gas and oxygen. The left ureter was exposed extraperitoneally and followed down to the bladder: this was made difficult by adhesions from a previous hysterectomy. The ureter was divided below a fusiform dilatation just above the bladder: the kidney was then exposed in the loin and removed with the ureter. The patient made a good recovery, and has had no further urinary trouble.

THE SPECIMEN (*Fig.* 256) consists of the left kidney and ureter. The kidney appears quite normal, its pelvis is of normal size or very slightly dilated. The ureter shows a normal smooth mucosa and no thickening of the muscle; at the lower end is a villous papillary tumour (*Fig.* 257) arising from the mucosa by a single narrow stalk. The villi are slightly pink, moderately opaque, and have a shining surface. The ureter is somewhat dilated at the level of the stalk of the tumour and for half an inch below this. Histological preparations (*Fig.* 258) show a papilloma with many villous processes of transitional epithelium covering a core of delicate connective tissue and capillaries with a few inflammatory cells. There is no suggestion of malignancy.

SUMMARY

A case is described of a benign papilloma of the ureter in a woman aged 42 years. Such tumours are even less common than the rare carcinomata of the ureter.

The photographs were taken by Mr. A. W. Collins, F.I.M.L.T.

REFERENCES

BARNES, R. W., and KAWAICHI, G. K. (1944), *Urol. cutan. Rev.*, **48**, 430.

COUNSELLER, V. S., COOK, E. N., and SEEFELD, P. H. (1944), *J. Urol.*, **51**, 606.

DAVY, R. (1884), *Brit. med. J.*, **2**, 757.

ILLINGWORTH, C. F. W., and DICK, B. M. (1941), *Textbook of Surgical Pathology*, 4th ed., 634. London.

JOHNSON, C. M., and SMITH, D. R. (1942), *J. Urol.*, **47**, 448.

LEBERT (1861), *Anat. Pathol.*, **1**, 269; **2**, 372 (quoted from MOORE).

MCMAHON, S. (1944), *J. Urol.*, **51**, 616.

MELICO, M. M., and FINDLAY, H. V. (1932), *Surg. Gynec. Obstet.*, **54**, 680.

MOORE, T. (1942), *Brit. J. Surg.*, **29**, 371.

MÜLLER, A. (1940), *Schweiz. med. Wschr.*, **70**, 232.

RAYER, F. F. O. (1841), *Traité des Maladies des Reins*. Paris. (Quoted from MOORE.)

CYSTOSCOPY IN THE DIAGNOSIS AND TREATMENT OF BILHARZIA HÆMATOBIUM INFECTION

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THE subject of bilharzia is of general interest now that many men in the Forces, who have served in East Africa and other tropical countries where such infection occurs, have returned home to Britain and the United States. Undoubtedly the appearances discussed below will be seen from time to time during cystoscopy in temperate climates and will cause some degree of difficulty in diagnosis to those who are not familiar with them.

In many cases a certain diagnosis and adequate treatment in *Bilharzia hæmatobium* infection necessitate the co-operation of physician and surgeon, or at any rate the employment of both medical and surgical procedures. Three years' experience in the Giriama Reserve, outside Mombasa, has forced me to the conclusion that to rely entirely on microscopical examination of the urine for bilharzia ova for the diagnosis of this condition is in many cases a delusion, and that it is fallacious to rely on the test of three negative urine examinations, after giving 24 gr. of antimony tartrate in a month, in deciding that the treatment has been adequate. I am convinced that cystoscopy is essential both to clinch the diagnosis and to assess the efficiency of the treatment given, and have tried to carry this out in as many cases as possible during the last two years on the coast of Kenya.

Some may think that the diagnosis of this disease by the use of the cystoscope requires an expert. In a sense that is true, but it takes very little time to become sufficiently acquainted with the signs of the disease to make a diagnosis even in early cases and to gauge the effect of treatment by comparing the picture seen before and after; this is especially so in areas where the disease is common. In examining the first few cases I was only able to record

what I saw without understanding the significance of my findings, but even after 10 to 15 cases the method was proving of definite assistance in the diagnosis of cases with a negative urine and in the differential diagnosis from other diseases of the bladder.

As far as is known, the effects of this disease are seen mainly in the bladder and to a lesser extent in the lower ureter and in the urethra. The *Bilharzia hæmatobium* worms live in the veins of the portal system, the female being considerably smaller than the male and living in a groove in the ventral surface of the male. When the female is ready to lay her eggs the worms migrate together against the venous blood-stream as far as they are able; the female then leaves the gynæcophoric canal of the male and travels still farther distally; when she has travelled as far distally as possible into the smaller venules she lays her eggs, the spines of which stick into the walls of the veins, thus preventing the blood-stream from sweeping them back towards the liver. In the majority of cases the worms reach first the hæmorrhoidal veins and then pass on to the veins around the lower ends of the ureters and those of the urinary bladder, prostate, and urethra; sometimes they pass on to other pelvic and perineal veins that anastomose with the hæmorrhoidals. Instinct seems to guide the worms to reach venules from where the eggs may easily reach the bladder and be voided in the urine. Thus it is that the earliest and most certain diagnosis of the condition can be made by examination of the bladder and that the effect of treatment can best be seen in this organ. Bilharziasis of the seminal vesicles, spermatic cord, epididymis, testes, and tunicæ vaginales is, I believe, commonly found in areas where this infection is prevalent, but even so the diagnosis is made much more simply by examination of the bladder. Even where other organs are affected, in nearly all cases the bladder is affected as well. In a few cases instinct seems to mislead the worms to pass from the portal veins to veins of the systemic system; this accounts for the fact that bilharzia, like syphilis, can produce symptoms in all the systems and mimic a great many other diseases. Bilharzia ova have been found at autopsy in nearly every organ of the body, but this does not alter the fact that the earliest and simplest method of diagnosis is made by cystoscopy.

Any one working in the Kenya Highlands, and I believe in Kavirondo where intestinal bilharzia is much more common, may well wonder whether a relatively elaborate method of diagnosis and of assessing treatment is warranted for a disease that is found, as it were, sporadically only. But in the Coast Province, especially in the native reserves near Mombasa, the disease is extremely common. In this area there are at least four native hospitals concerned. At a conservative estimate 90 per cent of the Wagiriama have been or are infected. Of 97 healthy Giriama schoolboys who were examined microscopically once only, 75 were found to have ova in the urine. Of 1500 out-patients examined once only at Kaloleni Hospital in 1943, 700 were found to have ova in the urine. Of these many more would have given a positive finding if examined repeatedly and many others would have shown stigmata at cystoscopy. There is no doubt in my mind that this disease, which leads to chronic anæmia, chronic cystitis, hydronephrosis, vesical calculus, vesical carcinoma, hepatomegaly, and splenomegaly, is to a very large extent responsible for the poor health and gross lack of initiative found amongst the Wagiriama. This is not only a problem of diagnosis and efficient treatment, but as much, if not more, of preventive medicine. When the water supply in the Giriama Reserve is improved and the people are instructed not to urinate into it we may expect *Bilharzia hæmatobium* to be much less common and the average health of the Wagiriama much improved. Even so, for years after the advent of adequate preventive medicine, we shall have many chronic cases to deal with, presenting greater difficulties in diagnosis and treatment than the relatively straightforward acute cases. If with the course of time the process of detribalization continues and African industries grow up in the larger towns, we may expect to be presented with these subacute and chronic cases in increasing numbers all over the country. For these reasons I believe that we shall need, in this Colony, more precise methods of diagnosis and more carefully controlled treatment than has been employed in the past.

Cystoscopy is not universally used in the various countries where the disease is prevalent. So far as I can ascertain it has not been employed in East Africa apart from work done by medical officers in the military hospitals during the last two or three years. In South Africa Cawston (1945) deprecates the employment of cystoscopy and believes that repeated examination of the last drops of urine will always give a positive finding in bilharzia infections. He has worked in Durban for many years and been closely associated with the treatment and prevention of this disease. Campbell Begg (1944), of Johannesburg, states his belief that " ova are so seldom seen in the urine in cases of chronic bilharzia that their absence has no diagnostic significance. The only reliable method of diagnosing the disease is by cystoscopy ". In Egypt I believe that all the larger hospitals employ this method, which is strongly recommended by Makar (1932), Professor of Surgery in Cairo University. In a report on one case of a young European who had been resident in South Africa, Christopherson and Ogier Ward (1934) describe the diagnosis and treatment. The treatment was controlled by cystoscopy performed before commencing the injections and one month after the last injection.

Cystoscopic Appearances.—The cystoscopic picture of bilharziasis of the vesical mucous membrane is characteristic. The mucosa is affected in the vast majority of cases. In cases where it is normal, changes in the muscle wall leading to fibrosis and calcification of the ova can often be detected by a straight radiograph of the bladder, as has been demonstrated by two workers in a hospital at Alexandria (Harlow, 1930). A bladder that can be felt per abdomen as a hard mass is, in my opinion, always carcinomatous and never merely a fibrosed and hypertrophied bilharzial bladder.

1. *Hyperæmia.*—The mucosal vessels become dilated, especially around the ureteric orifices, which are often surrounded by an area of œdema. This is the earliest change, but it is not specific.

2. *Tubercles.*—These are mostly found in the trigone or around the orifices, but may be seen anywhere; pale in colour, surrounded by a zone of hyperæmia, they vary in size from that of miliary tubercles to a good deal larger. (*Fig.* 259.)

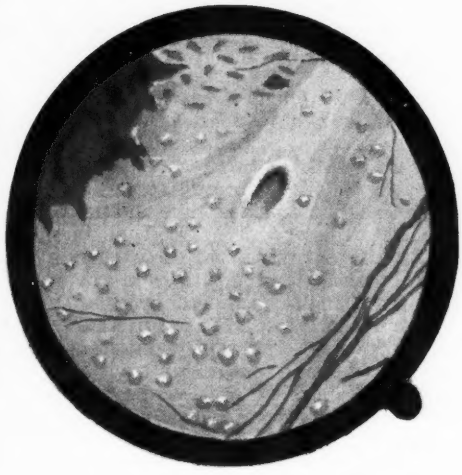

FIG. 259.—Case of bilharzial cystitis showing numerous tubercles around the left ureteric orifice, a small hæmorrhage above, and a group of dilated veins below.

3. *Closely-set Tubercles.*—These give the appearance of heaped-up granulations, often partly covered by blood-clot.

4. *Granulomatous Polypoid Growths.*—These are often found together with granulations; they have a broad base and are slightly lobulated and have an irregular knobly appearance.

5. *Bullous Cysts.*—These are pale colourless cysts twice the size of tubercles; sometimes they are solitary and sometimes found in clusters; they are due to epithelial hyperplasia which blocks the openings of the submucous glands and are akin to the Nabothian follicles found in chronic cervicitis. Sometimes these cysts are the only sign of bilharzial cystitis.

6. *Ulcers.*—These are usually shallow and small, but when secondarily infected may resemble an early carcinoma; they may be differentiated by watching the effect of antimony treatment.

7. *Chronic Bilharzial Cystitis.*—The mucosa is covered by a layer of thick mucus, giving a blurred and hazy appearance; in the mucus are suspended numerous glistening white pinhead calcareous particles.

We can now consider in greater detail specific types of case in which cystoscopy is of value in diagnosis and treatment.

DIAGNOSIS

1. No Ova found in the Urine.—In many cases of bilharziasis there are gross changes in the bladder wall and submucosa where no ova are being voided in the urine. The ova pass from the venule supplying the bladder into the muscle coats and submucous layer. Here they may become encysted and surrounded by fibrous tissue, and calcification occurs in some cases. Others in the submucous layer cause the formation of bilharzial tubercles which on cystoscopic examination are slightly larger than, but otherwise similar to, the tubercles seen in tuberculosis. These tubercles are formed of proliferating epithelial cells of the mucous membrane, in the midst of which may be found a few ova, and this change is presumably due to the irritation of the ova or their toxins. The presence of the characteristic tubercles is pathognomonic of bilharzial infection; in many cases they may be seen when repeated examination of the last few drops or urine to be passed has been negative. Only when the ova have ulcerated through the mucosa do we find them in the urine. In many of these cases there are visible shallow ulcers with a dark base due to coating with altered blood. The afore-mentioned changes occur most commonly in the trigone and around the ureteric orifices. In a few cases no changes are seen at the first examination beyond slight œdema around the orifices, with considerable injection around them and over the trigone. The characteristic symptom of pain at the tip of penis during micturition is present, presumably due to irritation of the trigone musculature by ova. In this case when ova are not present in the urine, examination will sooner or later reveal the presence of the characteristic bilharzial tubercles. In another small group of cases the tubercles may be seen though there are no symptoms relegated to the bladder.

Case 1.—S. G., male Giriama, aged 30. First seen in August, 1942, he had been treated one year previously for hæmaturia and pyuria of uncertain origin with sulphapyridine and antimony tartrate. On admission he complained of burning pain in the urethra during micturition. No gonococci or ova were found on repeated examination of the urine. Cystoscopy revealed only a little mucus all over the bladder, a few scattered blood-clots, and slight injection over the trigone. Cystoscopy one month later showed a few areas of hæmorrhage and a few small ulcers. Cystoscopy three months later showed only a few clots of blood on the bladder wall; he was then given a course of combined antimony tartrate and fouadin. He came back to hospital in 1944 complaining of burning pain during micturition and occasional hæmaturia. The urine was still negative. Cystoscopy revealed the presence of groups of transparent cysts and a few characteristic tubercles. He asserted that he had followed the prophylactic régime against contraction of this disease since the time of his first coming to hospital. Therefore, presumably, the first course of treatment was inadequate; definite tubercles were seen for the first time only two years after contracting the disease.

Case 2.—J. K., male Giriama, aged 55. Admitted to hospital in April, 1944, with a large swelling in the left loin. There were no symptoms referable to bladder or urethra, and there was no hæmaturia. Cystoscopy was performed in order to confirm that the tumour was renal in origin. The characteristic tubercles of bilharzia were seen around the orifices and in the fundus. The absence of symptoms, therefore, coincided with absence of trigonal irritation. On removal the tumour was proved to be a hypernephroma.

2. Other Conditions may resemble Bilharzia Infection or may be Concomitant with it.—The characteristic symptoms of vesical bilharziasis—pain during micturition, frequency, hæmaturia, and strangury—are common to a number of other diseases (Allen, 1936). For this reason it is advisable when ova are not discoverable in the urine

to cystoscope the patient in order to exclude the presence of some other disease. On the other hand, the presence in this hospital of numerous bladder scoops and stone-holding forceps which were passed on from the old hospital in Mombasa after the last war show that many cases of bilharzial cystitis were diagnosed as vesical calculus.

Case 3.—K. M., male Giriama, aged 40. Treated 1½ years previously for bilharzial cystitis, he now complains of frequency, burning pain during micturition, and pyuria, with attacks of strangury. Cystoscopy revealed the presence of a large calculus; the ureteric orifices were dilated to about ten times the normal size. There was marked trabeculation and two or three diverticula were present. This man died from uræmia when he might well have recovered had he been treated for the calculus when he first came to hospital.

Case 4.—B. K., male Giriama, aged 45. He was admitted to hospital complaining of suprapubic pain, frequency, and dysuria. The bladder was enlarged to 3 in. above the pubes and its wall was thick and hard; no ova were found in the urine. At operation the bladder was full of papillomatous new growth which on section proved to be carcinomatous. Clinically this was a typical case of long-standing bilharziasis with superadded chronic cystitis.

3. It is necessary to be on the look out for Sequelæ.—Cases of recent bilharzial infection are unlikely to show sequelæ, but other diseases of the bladder may be present at the same time; most long-standing cases of bilharziasis show sequelæ, as, for instance, polyps, papillomata, and carcinomata resulting from long-continued action of bilharzial prostatitis. For these reasons it is highly desirable that cystoscopy should be undertaken before the commencement of treatment, even when microscopy reveals the presence of ova in the urine (Makar, 1935).

Case 5.—K. M., male Giriama, aged 25. He was admitted with five months' history of hæmaturia, pain on micturition, and pain over the bladder. Examination of the urine showed the presence of ova, pus cells, and red blood-cells. Cystoscopy revealed the presence of a large irregular faceted calculus, covered with altered blood and a concomitant bilharzial cystitis with ulcers and tubercles.

Case 6.—N. K., male Giriama, aged 50. He was admitted to hospital with a right inguinal hernia. On examination the urine was found to be blood-stained; on rectal examination the prostate was considerably enlarged; ova were found in the urine. At cystoscopy there was intravesical enlargement of the prostate and a mild bilharzial cystitis as evidenced by the presence of the characteristic tubercles and slight injection of the trigone.

4. Often it is necessary to investigate the Ureters and the Renal Function.—Bilharziasis can affect any part of the bladder, but the trigone and area around the ureteric orifices are most commonly affected. The lower end of the ureter for one or two inches above the bladder is quite commonly diseased. The kidney and upper ureter are only secondarily affected as the result of prolonged back pressure and chronic infection.

In cases which present with attacks of renal colic, catheters should be passed along the ureters to demonstrate the presence or absence of obstruction in their lower ends. Where laboratory facilities are not available the renal function may be estimated by intravenous injection of indigocarmine, which is ejected from the orifices in seven to ten minutes when the renal function is good. Where X rays are not available the capacity of the renal pelvis can be estimated by injecting dilute mercury oxycyanide solution up the catheter until slight pain is felt in the loin.

Case 7.—C. K., male Giriama, aged 40. He was admitted with a two months' history of dysuria and strangury. No ova were found in the urine; he developed an attack of renal colic whilst in hospital. Cystoscopy revealed a slightly pouting left ureteric orifice with injection of the trigone and petechial hæmorrhages. The catheter only passed 1½ in. up the left ureter; no indigocarmine was seen emerging from the left ureter, though it emerged from the right in ten minutes. Intravenous peylography showed a block 1½ in. above the bladder in the left ureter. At operation 1 in. of thick stenosed ureter was removed from just above the bladder and the ureter transplanted into the bladder slightly higher up. Section showed that the removed portion of ureter was bilharzial.

TREATMENT

1. The efficient and successful treatment of *Bilharzia hæmatobium* infection is by no means as simple a matter as might be thought at first sight. The concentration of antimony tartrate required to kill the worms is not far short of that which proves severely toxic to the patient. Egyptian workers state that one case in two thousand dies from the toxic effects of this drug. Most of the cases treated in the Giriama Reserve were given 25 gr. of the drug within a period of four weeks, and nearly all of these showed toxic symptoms well before the end of the course. Valuable data can be gained as to the efficiency of treatment by comparison of the cystoscopic appearances before treatment with those two weeks after the completion of a course of treatment; where the treatment has been adequate cystoscopy will show marked improvement in any concomitant acute cystitis and marked retrogression of granulomatous polyps and bilharzial tubercles. It is recommended that the patient should return after three months and again after six months for examination, and if he still has symptoms or abnormal constituents in the urine, such as red cells or pus cells, he should be cystoscoped again. If ulcers, granulomata, or tubercles are present, or if there is a cystic cystitis, a further course of treatment should be given.

Case 8.—C. M., male Giriama, aged 20. He was admitted with a history of three months' hæmaturia and pain during micturition; many ova were found in the urine. Cystoscopy revealed numerous small ulcers and large blood-clots. The left ureteric orifice was slit-like and surrounded by pale grey nodular growth covered with mucus, in which were suspended numerous small calcareous particles. The right orifice was completely obscured by polypoid granulomata, which were covered with typical tubercles. One month later, after treatment, cystoscopy showed that the growth surrounding the right orifice was reduced in size by one-third. The granulomata obscuring the left orifice were reduced in size by three-quarters; no ulcers were seen; there were only scanty tubercles and the concomitant acute cystitis had subsided. The patient was symptom free.

Case 9.—M. K., male Giriama, aged 30. He was first seen in July, 1942, complaining of hæmaturia of many years' duration. Bilharzia ova were found in the urine. At cystoscopy an acute cystitis was revealed, the mucosa being bright red. The ureteric orifices were hidden by a large bright-red cauliflower mass (*Fig.* 260). He was given a course of antimony tartrate and fouadin. On March 2, 1943, he was symptom free. Cystoscopy showed a smooth undulating swelling of the whole of the trigone and ureteric bar, resembling hills and valleys. Both orifices were slit-like. There was a small sessile granular tumour immediately below the left orifice and a smaller but similar tumour a little above it; the whole mucosa was covered by a thin layer of mucus in which were suspended numerous small calcareous particles; no signs of active bilharziasis were seen.

FIG. 260.—*Case* 9. Showing large papillomatous bilharzial polyp in bladder. (Before treatment.)

On May 29, 1944, he was still symptom free and reported for routine cystoscopy. This showed a pale yellow mucosa covered with mucus in which were suspended calcareous particles as before. There were no signs of active bilharzia; the hill-and-valley appearance was much reduced, the orifices were still slit-like, and no signs of the previous granulomata could be detected (*Fig.* 261).

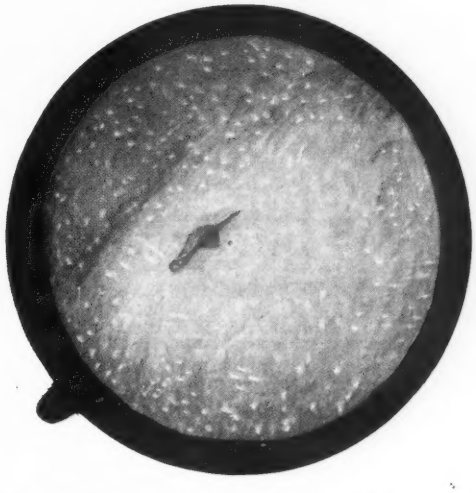

FIG. 261.—*Case* 9. After treatment: there is no vestige of the polyp. The mucosa is covered with mucus, in which are suspended numerous calcareous particles.

2. As pointed out by Makar (1942), the bilharzial toxins in the bladder wall, acting over a period of years, frequently lead to a carcinomatous change. To be at all effective, treatment must be undertaken as soon as possible after this change has set in. In the past three years I have seen at least six cases with a history of chronic bilharzia which came to hospital only when the bladders were full of masses of carcinomatous tissue; secondary invasion was widespread in all these cases. If seen at an earlier stage they might have been cured by transplantation of the ureters, followed by total cystectomy. The conclusion to be drawn is that routine cystoscopy should be performed at six-monthly intervals in all cases where there is permanent damage to the bladder wall or mucosa, as a result of bilharzia infection, in order that any carcinomatous change should be detected as soon as possible. Calcification in the bladder wall may be detected by X-ray examination. Permanent changes in the mucosa are evidenced by the finding of a covering of mucus all over the bladder, with calcareous particles suspended in it, at repeated routine cystoscopy. In *Case* 8 it was not possible to exclude bilharzial carcinoma until after the course of treatment, when the second cystoscopy showed marked retrogression of the granulomatous and polypoid growths. In *Case* 9 the cystoscopy carried out before treatment revealed signs that were very suspicious of carcinoma. This possibility was only ruled out when the second cystoscopy showed marked retrogression of the granulomata. The mucous and calcareous particles found to persist in a symptom-free patient two years later show that there was permanent damage to the mucosa, resulting in a chronic catarrhal state. This is the type of patient who should be cystoscoped at intervals of six months in view of the possibility of malignant change.

SEQUELÆ

The sequelæ of this disease present either more or less complicated problems in urogenital surgery. At present in the Giriama Reserve they are often seen.

1. Chronic Cystitis.—We have already discussed this condition.

2. Calculus Formation.—The calculi are mostly of calcium phosphate; they may be large or small, single, or multiple, and contain calcified ova.

3. Changes around the Ureteric Orifices.—Dilatation, scarring, obstruction, and hypertrophy all occur. These are among the most interesting changes seen in biharziasis and have a great number of individual characteristics.

4. Hypertrophy of the Bladder Musculature.—This leads to hydronephrosis and the usual accompanying renal changes. Renal failure would seem to be long delayed.

5. Renal Colic.—This is due to ureteral obstruction at the orifice or slightly above it.

6. Carcinoma.—This may assume various forms; it is more rapidly progressive than most non-bilharzial types. In Egypt bilharzial cancer is much more common than the non-bilharzial. There

seems little doubt that this type is due to the previous long-standing bilharzia infection; it has been ascribed to (*a*) long-standing sepsis, (*b*) mechanical irritation, and (*c*) prolonged action of toxins from the ova (Makar, 1942).

SUMMARY

1. Routine use of the cystoscope in both the diagnosis and treatment of bilharzial cystitis is advocated.

2. The typical appearances at cystoscopy are described.

3. A number of individual cases are presented, indicating in detail where cystoscopy can help in avoiding the difficulties of early and differential diagnosis and in assessing the efficacy of treatment.

4. The common sequelæ of this condition are enumerated and described briefly.

My thanks are due to the Hon. Director of Medical Services for permission to publish this paper.

REFERENCES

ALLEN, K. W. (1936), *E. Afr. med. J.*, Dec.
BEGG, R. CAMPBELL (1944), *S. Afr. med. J.*, July 22, 239.
CAWSTON, F. G., personal communication.
CHRISTOPHERSON, J. B., and WARD, R. OGIER (1934), *Brit. J. Surg.*, **21**, 84.
HARLOW, F. W., and AFIFI MAHMOUD (1930), *Urol. cutan. Rev.*, **34**, Sept.
MAKAR, NAQUIB (1935, a), *J. Egypt. med. Ass.*, Feb. 2.
— — (1935, b) *Soc. intern. Chir., Le Caire.*
— — (1942), *J. Egypt. med. Ass.*, **25**, 5, 6.

THYROTOXICOSIS AS A SURGICAL PROBLEM

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THE recent introduction of thiourea and thiouracil as therapeutic agents in thyrotoxicosis caused many physicians to believe that these compounds would displace surgery. Despite early optimistic reports on the efficacy of thiouracil, certain types of thyrotoxicosis appeared to remain essentially a surgical problem. These included thyrotoxicosis associated with heart failure, the more serious types of cardiac arrhythmias, a large goitre, and marked exophthalmos. As experience with these drugs has grown, there has been a tendency to view with less optimism their curative properties. This in a large degree has been due to their toxic properties, including such reactions as agranulocytosis, fever, dermatitis, jaundice, œdema, arthritis, purpura, lymphadenopathy, cardiac arrhythmia, and even an exacerbation of the thyrotoxicosis. Further, it is by no means clear that, in the large majority of cases, thiouracil can be discontinued without a recurrence of the symptoms of thyrotoxicosis. At the best, therefore, the thiouracil medication in thyrotoxicosis is not unlike the insulin therapy of diabetes—in each case the disease is controlled, not cured. Surgery, therefore, has certain advantages in that a cure of the thyrotoxicosis can be established quickly, after which no further controlled observations on the patient are necessary. It is probable that many patients with diabetes would prefer a surgical cure to insulin therapy. In discussions on the various methods of treating thyrotoxicosis, the chief criticism against surgery is its operative mortality. Until 1923 the operative mortality was sufficiently high, except among especially skilled surgeons, to make operation a hazardous procedure. In 1923 the action of iodine in thyrotoxicosis became more clearly understood as a result of observation on the basal metabolism. The operative mortality of the leading thyroid surgeons fell below 1 per cent, and operations for the relief of thyrotoxicosis were now attempted not without success by less experienced thyroid surgeons. Paradoxically there was a tendency for a rise in the mortality from operations for thyrotoxicosis in later years, but this was doubtless due to operations on patients who, before this, would have been too ill to withstand surgery.

In the paper which follows, which is based on a study of the last sixteen years, an attempt is made to follow in detail the effects of iodine on the basal metabolism in thyrotoxicosis, the value of iodine before and after subtotal thyroidectomy, the post-operative reaction, and the early assessment of a successful thyroid operation.

I. IODINE AND IODIDE DOSAGE IN THYROTOXICOSIS, AND ITS EFFECT ON THE BASAL METABOLISM AS A GUIDE TO OPERATION

HISTORICAL

Iodine Administration.—In 1813 Courtois discovered iodine. Prout (1834) maintained that in 1816 he successfully used an iodine compound in the treatment of goitre and that his remedy was adopted three years later by Dr. Elliotson of St. Thomas's Hospital. Prout did not publish these findings until 1834, by which time Coindet (1820) had already drawn attention to the value of iodine in simple and probably in toxic goitre. The credit, however, must go to Trousseau (1868) for describing clearly not only that iodine could bring about an artificial remission in the course of thyrotoxicosis, but also that the beneficial effects of iodine medication quickly passed off as soon as the drug was stopped. Trousseau's exact words are worth recording :—

In the course of October, 1863, I was consulted by a young married lady who habitually resides in Paris. She was suffering from a subacute exophthalmic goitre. The bronchocele was of great size. When I examined her for the first time, although I had let her rest for a long while and although I repeated the examination several times and at sufficiently distant intervals, so as to make

sure that she was no longer under the influence of emotion, I still found that her heart-beat was at the rate of 140–150 times in the minute. I recommended hydropathy and I wished to administer at the same time tincture of digitalis, but, preoccupied with the idea that there would be some danger in giving iodine, I wrote iodine instead of digitalis, so that the patient took some 15–20 drops of tincture of iodine a day for a fortnight. When she came back to see me her pulse was 90. I found out my mistake and I substituted tincture of digitalis for that of iodine, but after another fortnight the pulse had again gone up to 150, so that I at once returned to the iodine.

Notwithstanding these exceptional cases, however, bear in mind that iodine generally does harm in Graves' disease.

Later Cheadle (1869), Ohleman (1911), Marine and Lenhart (1911), and Waller (1914) confirmed Trousseau's observations, but the influence of Kocher and his school (1910, a, b), with their grave warning of the dangers of an iodine-precipitated thyrotoxicosis, dissuaded most clinicians from employing iodine therapeutically for the next ten to fifteen years. By 1920, when more simple forms of apparatus became available, measurement of the basal metabolism was being carried out more frequently, and at or about that time a series of publications appeared on the effects of iodine and its compounds in thyrotoxicosis, including studies of the basal metabolism. These were contributed by Neisser (1920), Loewy and Zondek (1921), Jagič and Spengler (1924), Cowell and Mellanby (1924), Starr, Walcott, Segall, and Means (1924), and Plummer and Boothby (1924). The greatest credit, however, must be given to Plummer and Boothby, for they clearly demonstrated the beneficial effects of iodine as a pre-operative measure. It is interesting to note that Plummer, in his first communication on this subject (1923), advised the administration of compound tincture of iodine *for ten days after operations* on the thyroid. Later, with Boothby (1924), he stated the properties of iodine more clearly. They pointed out that in thyrotoxicosis the maximum improvement usually occurred after iodine, in adequate doses of at least 10 min. daily, had been taken for eight to ten days. They also drew attention to the fact that the maximum improvement might be delayed for two or even three weeks. They therefore recommended that any operation on the thyroid should be postponed until it was established that no further improvement could be obtained from the iodine. Such a procedure brought about a fall in the mortality-rate of thyroidectomy for the relief of thyrotoxicosis to less than 1 per cent at the Mayo Clinic.

The Common Action of Iodine and Its Compounds.—It is not clear how iodine exerts its action on the thyroid gland, but it has been established that not only free iodine but all its compounds are alike in producing clinical changes in thyrotoxicosis. Pure iodine has been used by Marine and Lenhart (1911) and Starr, Walcott, Segall, and Means (1924); potassium iodide by Neisser (1920), Loewy and Zondek (1921), and Cowell and Mellanby (1924); sodium iodide by Jagič and Spengler (1924); tincture of iodine (2·5 per cent iodine, 1·5 per cent potassium iodide in alcohol) by Trousseau (1868) and Waller (1914); hydriodic acid by Fitzgerald (1926); Sajodin (calcium iodobehenate) by Fulton and Ault (1930); and Lugol's solution (5 per cent iodine, 10 per cent potassium iodide in water) by Plummer (1923), Starr, Walcott, Segall, and Means (1924), and many others. In addition to the oral administration, iodine has also been given with benefit by inhalation as ethyl iodide (Lerman and Means, 1931). Reference is also made in general text-books to the successful administration of iodine intravenously, rectally, or simply by skin application. It is clear, therefore, that iodine is rapidly absorbed through the skin or through any mucous membrane, and that iodine or any of its salts are equally effective. There are several publications, among others by Fitzgerald (1926) and Fulton and Ault (1930), showing clearly that the form in which iodine is administered is of little consequence. The most popular form in which iodine is given is Lugol's solution, and this is doubtless due to the influence of Plummer, who chose this mixture "because it was an aqueous solution of iodine and provided a large amount of iodine loosely combined with potassium". It is interesting to note that Lugol described the preparation of this solution in 1830 as a cure for tuberculous infections. His memoir in an abstracted form was later translated into English by Markwick (1846).

Dosage of Iodine.—There is still some controversy about the ideal or proper dosage of iodine in thyrotoxicosis. The consensus of opinion would appear to be that for the prolonged treatment of thyrotoxicosis by iodine alone, the dosage should be small and in the region of 6–12 mg. of iodine daily (1–2 drops of Lugol's solution), but that as a pre-operative measure the dosage should be much larger. As this paper is concerned with the preparation of patients for surgery, this will now be considered. It is not clear why such large doses of iodine are necessary or advisable as a pre-operative measure. Although Thompson, Bailey, Thompson, and Thorp (1930) maintained that the minimum amount of iodine that will produce the maximum reduction in the basal metabolism is not greater than 6 mg. of iodine daily (1 drop of Lugol's solution), they have stated for no apparent reason that five times this amount should be given daily before operation. Plummer and Boothby (1924) go further and state that a minimum of ten drops of Lugol's solution is desirable, as they observed certain patients who did not react to five drops but did to ten drops daily. It was also their experience that an increased dosage of ten drops three times per day produced better and quicker responses. Both Neisser (1920) and Loewy and Zondek (1921) appeared to have introduced the scheme of increasing the daily dosage of iodine from fifteen drops of potassium iodide solution to an optimum of sixty drops. This system has been advocated by Joll (1932), who started with five drops of Lugol's solution three times per day and increased the amount by three drops per day until the dosage was thirty to forty-five drops per day a few days before the operation.

It was with a view to clarifying the various actions of iodine on the basal metabolism in thyrotoxicosis that the following experiments were designed. In view of conflicting reports, a critical inquiry was made into iodine dosage and the types of iodine therapy used as pre-operative measures.

EXPERIMENTAL

In *Table I* are given a list of solutions and salts of iodine in common use, with details regarding their composition and the amount of total iodine. As all forms of iodine are equally effective it can be

Table I.—SOLUTIONS AND SALTS OF IODINE, WITH COMPOSITION AND AMOUNT OF TOTAL IODINE

IODINE COMPOUND	FREE IODINE	SALT OF IODINE	VEHICLE	TOTAL IODINE (FREE IODINE PLUS IODINE IN COMBINATION)
				1 min. contains
Liquid:—				
1. Liq. iodi mitis (B.P., 1932) or Tinct. iodine	2·5 per cent	Potassium iodide, 1·5 per cent	Alcohol	2·16 mg.
2. Liq. iodi simplex (B.P., 1932) or Tinct. iodi (French Codex of 1908)	9·0 per cent	Nil	Alcohol	5·33 mg.
3. Lugol's solution (B.P., 1936)	5·0 per cent	Potassium iodide, 10 per cent	Water	7·49 mg.
4. Hydriodic acid	—	Hydrogen iodide, 10 per cent	Water	5·89 mg.

		1 gr. contains
Solid:	Potassium iodide	49·6 mg.
	Sodium iodide	54·9 mg.
	Ethyl iodide	52·9 mg.
	Sajodin (calcium iodobehenate)	15·6 mg.

calculated from *Table I* that 30 minims of Lugol's solution are roughly equivalent to $4\frac{1}{2}$ gr. of potassium iodide and 4 gr. of sodium iodide each containing approximately 220 mg. of iodine. In the results which follow iodine was given in amounts of 180 to 200 mg. daily in the form of pure iodine, Lugol's solution, or potassium iodide, but, as previously explained, these different forms of iodine have no significance.

The basal metabolism was measured by the closed-circuit method, the accuracy of which has been reported (Robertson, 1937). The apparatus used was the Benedict-Roth with recording kymograph. All machines were alcohol-checked (Barrett and Robertson, 1937) and found accurate, but in addition each was checked once a week by a model whose basal metabolism is in the region of 56 calories per hour. All subjects studied were in-patients, but the technique of measuring their basal metabolism was essentially the same as that described for out-patients (Robertson, 1944). The subjects were brought to a room in the Courtauld Institute with a temperature of 20–23° C. from the wards in chairs on at least two successive mornings under basal conditions. If, on the second day, the duplicates agreed within 5 per cent (and were lower than the first-day readings) the lower reading was taken as the basal metabolism. On the other hand, if the duplicate readings were not within 5 per cent, or the second readings were higher than those on the first day, then readings were taken on subsequent days until a constant reading was obtained. Such difficulties were experienced in 5 per cent of cases. After the initial reading had been determined, a further reading was taken in ten to fourteen days and thereafter daily or on alternate days until a constant reading was obtained after simply rest and sedatives. Then 180–200 mg. of iodine was given daily, and the basal metabolism again measured daily or on alternate days after the seventh day of iodine therapy, until or unless a constant reading was obtained. Charts 1–5 (*Figs.* 262–266) show observations on the basal metabolism in five selected cases of thyrotoxicosis, illustrating how the action of iodine can vary from case to case and in the same case from time to time. In Chart 6 (*Fig.* 267) the different reactions of the basal metabolism in thyrotoxicosis to iodine is given diagrammatically.

In the series of cases investigated it was found that simple rest in bed and sedatives brought about a fall of 15 to 40 per cent in the initial basal metabolism.

Chart 1 (*Fig.* 262).—In thirteen days the maximum reduction in the basal metabolism took place, and operation was carried out on the seventeenth day of iodine therapy. The post-operative basal

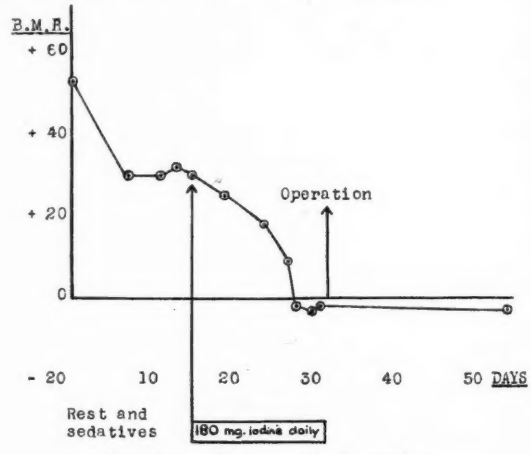


FIG. 262.—Chart 1. M. C., aged 35.

metabolism was not significantly different from the lowest basal metabolism after iodine. This is the most satisfactory type of iodine response, in which is produced a maximum fall in basal metabolism to the patient's normal level.

Chart 2 (*Fig.* 263).— Iodine brought about a fall in the basal metabolism to the region of + 30, and no further improvement took place. Iodine was unable to control the thyrotoxicosis further. Successful subtotal thyroidectomy was carried out with a post-operative basal metabolism of −9 and well below the lowest reading on iodine.

Chart 3 (*Fig.* 264).—The first course of iodine produced a curve similar to that in Chart 2. Instead of operating with a basal metabolism in the region of + 30, it was decided to stop the iodine for a period of two months and to give the patient sedatives. At the end of two months iodine produced a marked fall in the basal metabolism to −3, at which level a successful subtotal thyroidectomy was carried out.

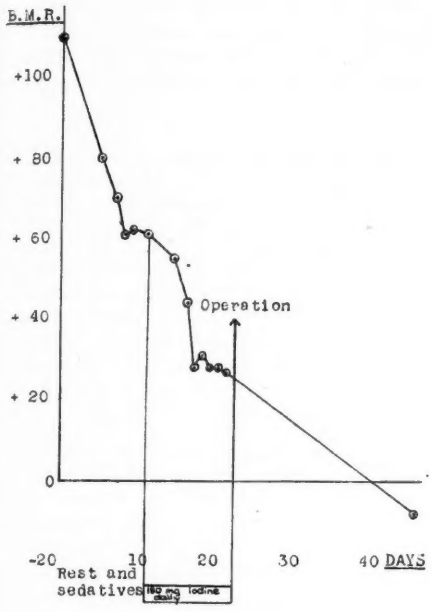


FIG. 263.—Chart 2. E. C., aged 42.

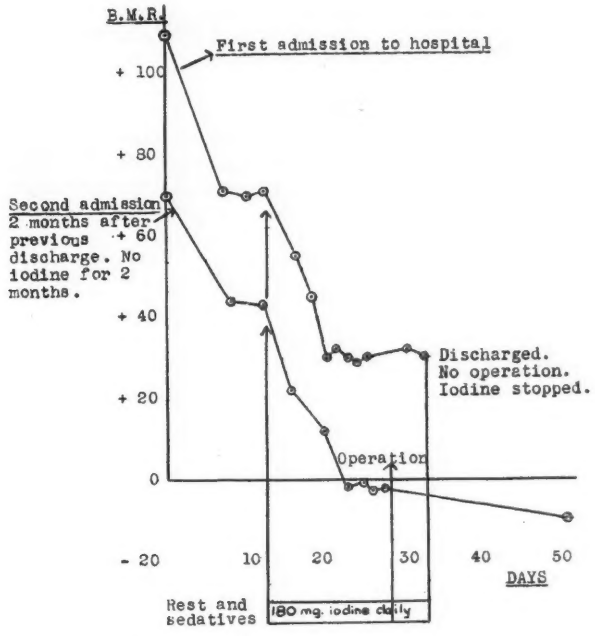


FIG. 264.—Chart 3. W. D., aged 28.

Chart 4 (*Fig.* 265).—This shows a most interesting response of the basal metabolism to iodine.

Days of Iodine Therapy	*Basal Metabolism*
None	+ 50
9	− 5
10	− 13
11	0
14	+ 8
15	+ 15
18	+ 22
21	+ 40

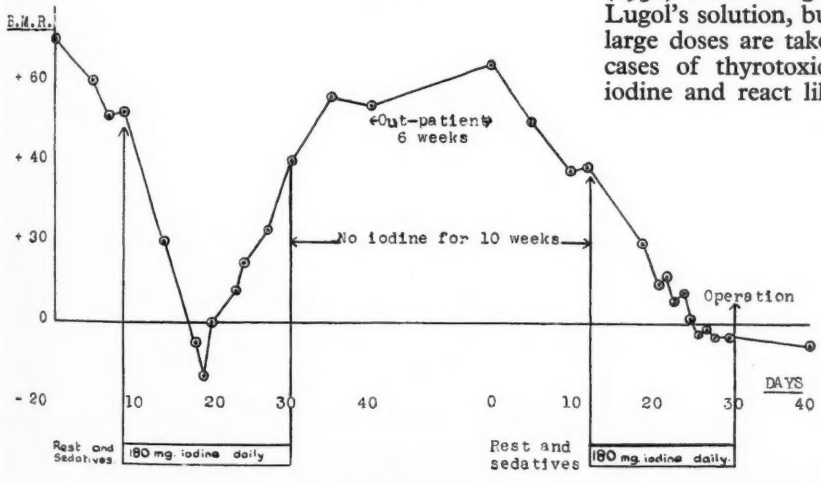


FIG. 265.—Chart 4. L. H., aged 23.

Iodine was now replaced by sedatives for six weeks and the basal metabolism rose to + 60. The next course of iodine brought about a steady fall in the basal metabolism to a plateau in the region of −3, at which stage a subtotal thyroidectomy resulted in a post-operative reading of −5. This case presented many interesting features. It showed the value of carrying out not isolated, but serial, basal metabolism determinations. Thus, if after the initial reading of + 50, further single readings were taken on the eleventh or the fourteenth day of iodine therapy the results of 0 or + 8 would have created the erroneous impression that the basal metabolism was falling, when, in fact, it was 'on the rise'. This is an example where a case becomes 'refractory' to iodine, and the drug loses its power to control the thyrotoxicosis. The condition has been well described by Thompson and Thompson (1931) when using small doses equal to 1 minim of Lugol's solution, but it also appears to develop when large doses are taken. It is important to recognize cases of thyrotoxicosis that become refractory to iodine and react like the first curve in Chart 4, as operation, if carried out when the metabolism is rising, is invariably accompanied by a thyroid crisis.

Chart 5 (*Fig.* 266).—Iodine caused an increase in the severity of the disease as shown by a rising metabolism from + 30 to + 45 in twenty-five days. Iodine was stopped for a month. The second course was followed by a steady fall in the basal metabolism to a plateau in the region of + 10, at which level a subtotal thyroidectomy was carried out resulting in a post-operative reading of −5. This case is unusual in that an active case of thyrotoxicosis was made clinically worse with iodine. It has already been reported by Kocher (1910) and Plummer and Boothby (1924), that adenomatous goitres without hyperthyroidism could be rendered toxic by the administration of iodine.

Chart 6 (*Fig.* 267).—This chart shows diagrammatically the different responses of the basal metabolism to iodine. From the point of view of operative

risk, the first two reactions are the most satisfactory. With the other two reactions with a rising metabolism the operative risk may be great.

DISCUSSION

It is difficult to understand why Lugol's solution should be the most popular form in which iodine is taken. It has a most objectionable burning taste,

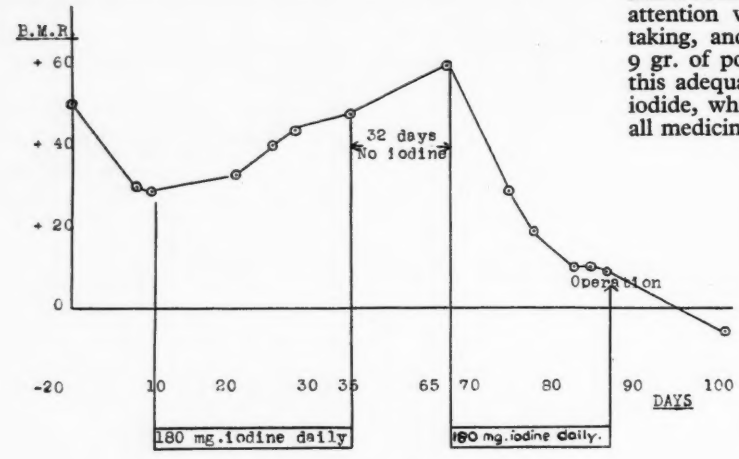


FIG. 266.—Chart 5. J. M., aged 50.

and the custom of giving it in drops is far from accurate, unless the solution is diluted to teaspoonful or other reproducible types of dosage. It is probable that the influence of Plummer, who chose this solution because it contained a large amount of dose of iodine) is equivalent to 4½ gr. of potassium iodide and 4 gr. of sodium iodide each containing approximately 220 mg. of iodine. It is important to realize that all soluble compounds of iodine are equally effective, as the following experience will demonstrate :—

A case of obvious thyrotoxicosis with exophthalmos had a basal metabolism of + 4. In reporting this result, attention was drawn to the fact that the patient was taking, and had taken for the previous fourteen days, 9 gr. of potassium iodide in a cough mixture. Despite this adequate control of the thyrotoxicosis by potassium iodide, when subtotal thyroidectomy was decided upon, all medicines were stopped and the patient given 15 min. of Lugol's solution daily for ten days before operation. This case, therefore, for fourteen days was having 448·4 mg. of iodine as potassium iodide, with the thyrotoxicosis controlled ; yet, because of a rule-of-thumb routine, Lugol's solution was given for ten days before operation, providing only 102·4 mg. of iodine, or less than a quarter the patient was receiving on potassium iodide.

There appear to be many advantages in employing as a pre-operative measure iodine in the form of potassium or sodium iodide. These medicines are not unpleasant and small doses of 2–2½ gr. per day (equivalent to 15 min. of Lugol's solution) are adequate in the preparation of cases of thyrotoxicosis for operation. On the other hand Lugol's solution, at present the most popular of iodine mixtures, lacks the advantage of simple iodides. It has been established that the minimum dose of iodine

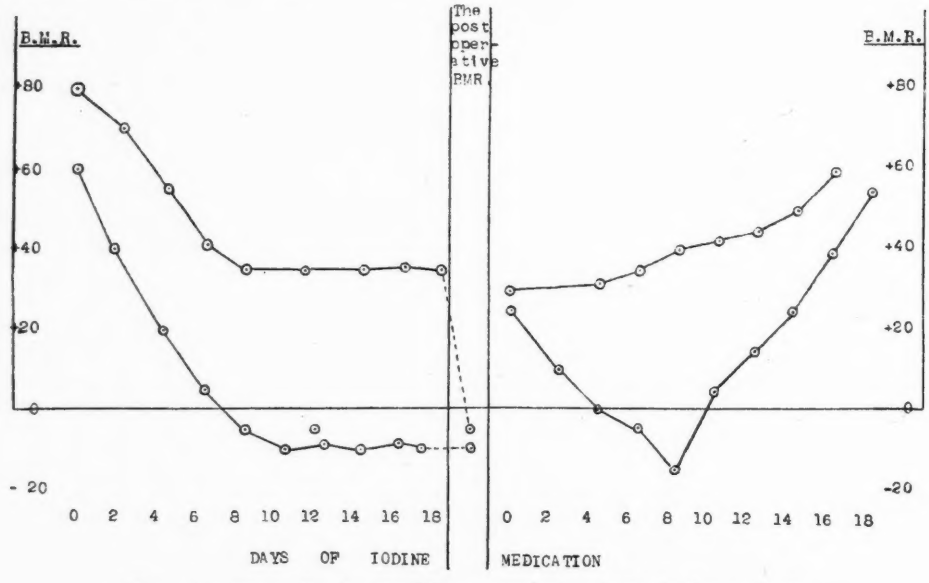


FIG. 267.—Charts showing the different reactions to iodine in thyrotoxicosis.

aqueous iodine, persists. Plummer may not have been aware that all salts of iodine were equally effective, and that the ability of iodine compounds to reduce the basal metabolism in thyrotoxicosis does not depend on the presence of free iodine, but soluble iodide. It has been shown that 30 min. of Lugol's solution (a common pre-operative daily that will produce the greatest clinical improvement and maximum reduction in the basal metabolism in thyrotoxicosis is in the region of 6 mg., or about 1 min. of Lugol's solution. As a pre-operative measure, iodine in excess of minimum requirements has been advocated, and doses from 5–30 min. per day are employed in various clinics. There is a

practice among some surgeons to give increasing doses of iodine, starting with 3 min. three times daily and working up to 10–12 min. thrice daily on the day before operation. This complicated system of iodine dosage is not governed by an experimental or scientific reasoning, and has nothing to commend it. It is, in fact, an undesirable procedure, for small doses of iodine can inhibit the maximum response of large doses. It would seem reasonable to commence and continue with large standard doses of 2-4 gr. of sodium or potassium iodide, or 15–30 min. of Lugol's solution, daily until the subject is ready for operation.

It has been stated by several investigators that the maximum benefit from iodine in thyrotoxicosis usually occurred after eight to ten days of medication. It has, however, been emphasized that this maximum improvement might be delayed two or even three weeks, and that it was inadvisable to operate until it was clear that no further improvement could be obtained from the iodine, even if this involved a delay of several weeks. Unfortunately, instead of adopting this sound principle first elaborated by Plummer and his colleagues of the Mayo Clinic, it is customary in certain clinics to replace this technique by a rule-of-thumb procedure whereby the operation takes place after ten days of iodine medication, irrespective of whether the maximum improvement has taken place or not. This has resulted, not uncommonly, in severe post-operative reactions. In assessing the maximum clinical improvement of a case under iodine, the basal metabolism is probably the best single guide. It is appreciated, of course, that a surgeon with much experience in thyroid surgery can by his personal judgement form an accurate opinion of the clinical state of a case of thyrotoxicosis. But all surgeons are not so fortunate, and for those without that experience, or those in the process of acquiring it, the basal metabolism can give a very accurate index of the iodine-control of the thyrotoxicosis, and the ability of the patient to withstand operation. In the present study it was found that, after due allowance had been made for rest and hospitalization, the basal metabolism fell to its lowest level with iodine in a period varying from six to twenty-five days. If patients are given iodine immediately on admission to hospital it seems probable that iodine will take longer than the above range to exert its maximum control. The danger in operating is not so much in delaying it, as in carrying it out too early during iodine therapy. It is important to realize that all cases of thyrotoxicosis do not react alike to iodine, nor any one case at different times. Iodine appeared to cause four well-defined reactions in thyrotoxicosis: (1) A fall in the metabolism to a low plateau which equalled or was not significantly different from the subject's normal basal metabolism obtained after a successful operation; (2) A fall in the basal metabolism to a plateau which is significantly higher than the basal metabolism after a successful operation; (3) A sharp fall in the basal metabolism, followed immediately by as sharp a rise; (4) A rise in the basal metabolism. The ideal pre-operative condition of the patient is one in which the basal metabolism is reduced by iodine to as low a level as possible and at the same time constant. The second condition is easier to achieve than the first, with the result that many operations are performed on patients with a constant basal metabolism, yet one that is raised well above normal. In these types of cases it is sometimes advisable to postpone operation until a more favourable response to iodine is obtained. Operation is contra-indicated when the basal metabolism is rising, a condition in which the patient is refractory to iodine. When it is considered that operation is not without risk for any of the above reasons, iodine should be stopped and replaced by sedatives such as quinine hydrobromide and luminal and a high-calorie diet, including glucose up to 6 oz. daily for four to six weeks. After iodine has been omitted for from four to six weeks, the thyrotoxicosis reverts to its primary untreated condition, and iodine again is able to bring about an artificial remission in the disease. It has erroneously been stated that the first iodine remission is the best. This is incorrect. Provided a period of four to six weeks of freedom from iodine medication is allowed to elapse, then iodine will continue to exert its control on the thyrotoxicosis.

In interpreting observations on the basal metabolism in thyrotoxicosis, it is important not to rely on isolated readings, particularly in the case whose basal metabolism suddenly falls, then rises, on iodine. Several readings at daily or two-day intervals will indicate how a case is reacting to iodine, whereas a single reading on the twelfth day would mislead. Thus, by the twelfth day of iodine medication the basal metabolism might, after a sharp fall, be 'on the rise', yet the figure, being lower than the initial reading, would erroneously imply that the basal metabolism was falling.

In this section a study has been made of the basal metabolism in thyrotoxicosis, and the relationship between the various reactions of the basal metabolism to iodine and the operability of the case. It is, of course, appreciated that there are other factors of importance to guide the surgeon in performing a successful subtotal thyroidectomy—such as the skill of the surgeon, his decision on the extent of surgery the patient's condition permits, the type of operation, the pre-operative treatment excluding iodine, the anæsthesia, and the immediate post-operative treatment. With these very important aspects of surgery of the thyroid gland, this paper is not concerned. It is believed, however, that one, if not the most important single assessment of a patient's ability to withstand a thyroid operation for thyrotoxicosis is the response to, and the level of the basal metabolism after, iodine medication. So-called thyroid surgeons, before and after the value of iodine was appreciated, have used their own judgement in deciding when to operate and their mortality-rates were low. Less experienced surgeons were not so fortunate. It is for the latter type of surgeon that the basal metabolism, as a means of assessing the ability of patients to withstand operation, can be of invaluable help.

SUMMARY AND CONCLUSIONS

1. Iodine and all its soluble compounds are equally effective in controlling thyrotoxicosis. The total amount of iodine in 30 min. of Lugol's solution (5 per cent iodine, 10 per cent potassium iodide in

water) is equivalent to that in 4½ gr. of potassium iodide and 4 gr. of sodium iodide each containing approximately 220 mg.

2. The disadvantages of Lugol's solution far outweigh its advantages as the ideal iodine compound in the treatment of thyrotoxicosis. The iodides of sodium or potassium are more satisfactory.

3. The system, which at present prevails in some clinics, of giving gradually increasing doses of iodine, has no justification. This unnecessary complication in dosage only confuses medical treatment, and it has no advantages over a constant daily dose of about 200 mg. of iodine.

4. The basal metabolism is probably the best single guide in assessing when a case of thyrotoxicosis is ready for operation. Surgeons experienced in thyroid surgery can rely, and have relied, on their clinical judgement in place of measurement of the basal metabolism, but for less experienced surgeons measurement of the basal metabolism provides a ready means of assessing the clinical state of the patient at any time during iodine medication.

5. The rule-of-thumb procedure of operating on cases of thyrotoxicosis after ten days of iodine medication is not without risk. The reason is that the maximum improvement obtained from iodine does not always occur on the tenth day—it may be earlier and it may be later. By the tenth day of iodine medication iodine may have lost its ability to control the thyrotoxicosis, and with the basal metabolism rising operation is not without risk.

6. Iodine causes four different types of reaction on the basal metabolism in thyrotoxicosis, and in two of the types operation on the thyroid is contra-indicated. These types of reactions are best detected by carrying out several basal metabolism determinations throughout iodine therapy. An isolated reading on the tenth or twelfth day of iodine medication, common in certain clinics, may give misleading information. Thus, by the tenth or twelfth day, the iodine may have lost its ability to control the thyrotoxicosis and the basal metabolism started to rise. At this point a reading would be lower than the initial basal metabolism, implying that the basal metabolism was falling.

7. The belief that iodine will produce its best response when given for the first time is incorrect. Provided an interval of four to six weeks has elapsed, when no iodine or its compounds have been taken, it will bring about its typical remision of the thyrotoxicosis again.

II. THE POST-OPERATIVE REACTION AFTER SUBTOTAL THYROIDECTOMY FOR THE RELIEF OF THYROTOXICOSIS

In the previous section it was stated that it seemed desirable to depress the basal metabolism to as low a figure as possible with iodine before operating on the thyroid. In the description of the cases that follow an attempt is made to prove that this procedure is justified. The proof is based on measurements of the basal metabolism immediately after subtotal thyroidectomy on cases where iodine brought about a varied response pre-operatively. In some cases iodine succeeded in causing the basal metabolism to fall to within normal limits, but in others iodine only brought about a slight fall and operation was carried out when the basal metabolism was at a high level.

Observations on the basal metabolism in cases of thyrotoxicosis immediately after operation have been reported by Sturgis (1923), Boothby (1921), Eberts (1921), and Segall and Means (1924). In this paper the findings of the above authors have been extended to include not only thyrotoxicosis, but also non-toxic goitre, simple glands of neck, and abdominal operations in an attempt to determine whether the post-operative rise in the basal metabolism is specific for a thyroid that is producing a toxic secretion.

METHODS

All metabolism determinations were carried out under strictly basal conditions. The pre-operative determinations were measured in a room in the Courtauld Institute where the patients were brought from the wards in chairs. In cases of thyrotoxicosis the first basal metabolism plotted on the chart was the reading taken after rest and sedatives had taken effect and was 15 to 40 per cent lower than the initial observation. After a constant reading was obtained 180 to 200 mg. of iodine were given daily in the form of potassium or sodium iodide or Lugol's solution. The second basal metabolism determination charted was the lowest constant reading reached after iodine, and this occurred, as stated in the previous paper, in from six to twenty-five days. The post-operative readings were taken in the wards at the bedside behind screens. For the first two mornings after operation the patients complained of difficulty in swallowing and painful necks, but the discomfort was not such as to prevent them having their basal metabolism measured. When fever was present, and this commonly occurred after operation, the necessary correction in the basal metabolism by subtracting 7·2 per cent for each degree F. was made as suggested by Dubois (1936).

In taking the temperature the thermometer was left in the mouth for six minutes and the readings were taken before those of the basal metabolism.

Pulse-rate readings of a minute's duration were taken at intervals during the metabolism determination, but only the lowest rate was recorded.

Blood-pressures were taken at the conclusion of the metabolism determination, and while the patient was still at rest.

In Chart 7 (*Fig.* 268) are the pre- and post-operative records of the basal metabolism in three selected cases of thyrotoxicosis showing different responses to iodine. In 'J.D.' the basal metabolism could not be depressed below + 50, in 'A.C.' it fell to the lower figure of + 24, while in 'M.D.' it fell to within normal limits. On the day after operation there was a rise in the basal metabolism in all three cases, the rise being greatest (47 per cent) where the pre-operative metabolism on iodine was the highest (i.e., J.D. at + 50), and only 15 per cent where iodine had caused the metabolism to fall to normal (i.e., M.D. at − 2). The fall in metabolism from the peak on the first day after operation was rapid, reaching normal limits in three to nine days, the return to normal being most rapid in the case where iodine had reduced the basal metabolism to

normal before operation. The basal pulse-rates recorded on Chart 8 (*Fig.* 269) followed the curves of the basal metabolism closely.

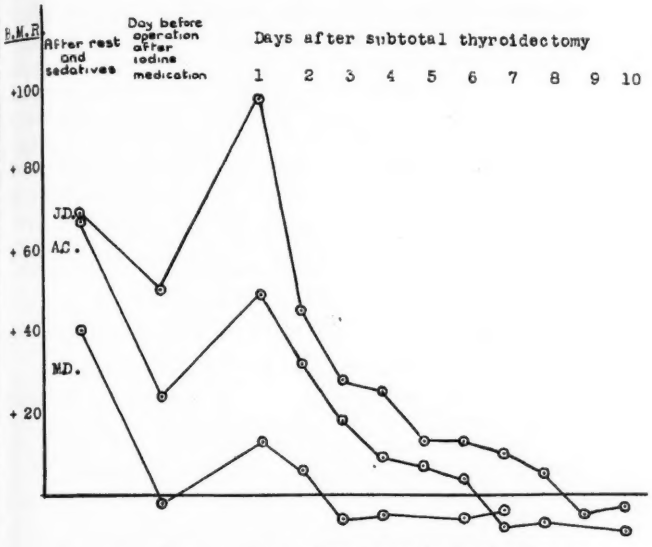


FIG. 268.—Chart 7. Pre- and post-operative basal metabolic rate records in three cases of thyrotoxicosis, showing different responses to iodine.

It is commonly believed that the post-operative reaction after subtotal thyroidectomy for the relief of thyrotoxicosis is indicative of a pouring out of a toxic secretion from the thyroid gland into the general circulation. This apparently is supported by the findings in Chart 7, which showed that the post-operative reaction is most marked in the case with the highest pre-operative basal metabolism after

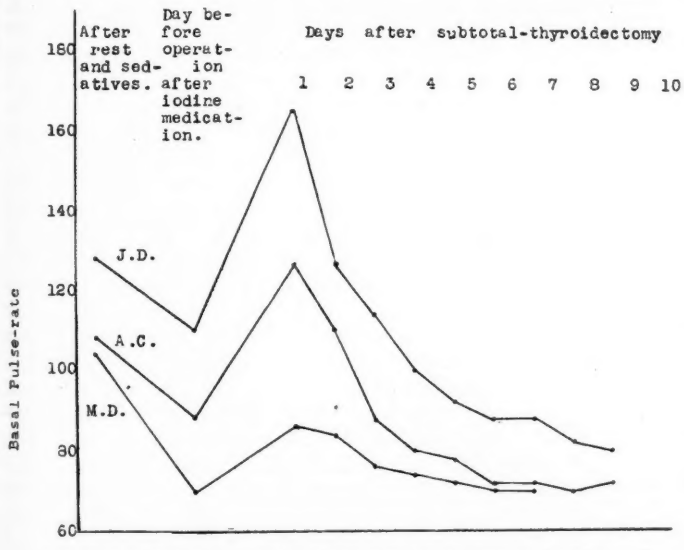


FIG. 269.—Chart 8. Basal pulse-rates in same cases as in Chart 7, showing the close approximation to the basal metabolism curves.

iodine medication, i.e., the most severe grade of thyrotoxicosis. In Chart 9 (*Fig.* 270) the post-operative curve of the basal metabolism in a case of thyrotoxicosis, reduced to normal with iodine, is compared with the curves following subtotal thyroidectomy for non-toxic goitre, and after operations for secondary glands in the neck and chronic appendicitis. It will be seen that all curves are

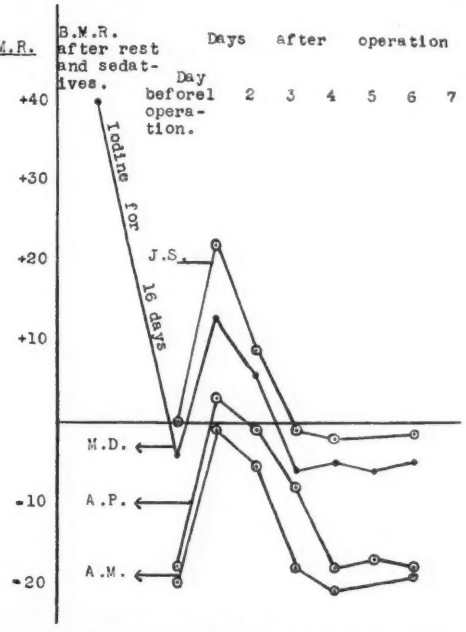


FIG. 270.—Chart 9. Comparison of post-operative curve of basal metabolism in a case of thyrotoxicosis reduced to normal with iodine (M. D.), with those of J. S., Subtotal thyroidectomy for relief of non-toxic goitre; A. P., For chronic appendicitis; A. M., For removal of secondary glands of neck.

comparable both as regards the percentage rise in metabolism of the peak on the day after operation, and the length of time the metabolism takes to return to normal. The pulse-rates are not charted, but they again followed the course of the metabolic rates closely. The post-operative rise in the basal metabolism is therefore not specific for thyrotoxicosis, but it occurs to the same degree after operations for the relief of non-toxic goitre, glands in neck, and chronic appendicitis.

Attention has been drawn by Lerman (1930) and Means (1937) to a temperature reaction of several days' duration which invariably accompanies the convalescence following subtotal thyroidectomy for thyrotoxicosis. In Chart 9 the daily temperatures after operations for the relief of thyrotoxicosis, non-toxic goitre, and chronic appendicitis are plotted. The findings of Lerman and Means are confirmed that fever, not associated with any evidence of infection, is present for several days after operations for the relief of thyrotoxicosis. This post-operative temperature reaction does not appear to be specific for operations on toxic goitres, as it also is present after operations on non-toxic goitre, for chronic appendicitis, and for removal of secondary glands in neck (not charted in Chart 10 (*Fig.* 271) in order to avoid confusing the other three curves).

In Chart 11 (*Fig.* 272) variations in the systolic blood-pressure after the various types of operations

are plotted. It will be seen that the blood-pressure rises after operations but returns to normal in two to three days, and that the elevations in blood-pressure are of the same degree irrespective of the nature of the operation.

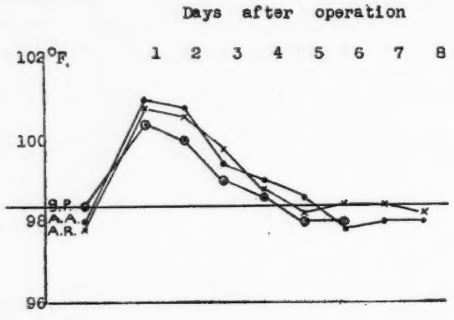


FIG. 271.—Chart 10. Temperature recordings after operations for the relief of thyrotoxicosis (A. A.), non-toxic goitre (A. R.), and chronic appendicitis (G. P.)

In the operations described above for the relief of the thyrotoxicosis, non-toxic goitre, appendicitis, glands of neck, and the like, various types of anæsthesia were employed such as the different forms of local, general, and local plus general.

Despite the use of different anæsthetics, the post-operative metabolisms appeared to be dependent only on the level of the pre-operative basal metabolism and unaffected by the type of anæsthesia employed. A study of the effects of a general anæsthetic on the basal metabolism was made, however, and the findings are shown in Chart 12 (*Fig.* 273). The patients were given an anæsthetic to allow of a simple gynæcological examination or an insufflation of the Fallopian tubes, but no surgical procedure involving trauma or incisions was carried out. In no case was the basal metabolism, in the days following the anæsthetic, above that of the pre-operative level. Observations on the pulse-rate and systolic blood-pressure also showed no significant change. It was, however, noted that it was not uncommon to find a slight fever of 99° F. on the day following the administration of the anæsthetic.

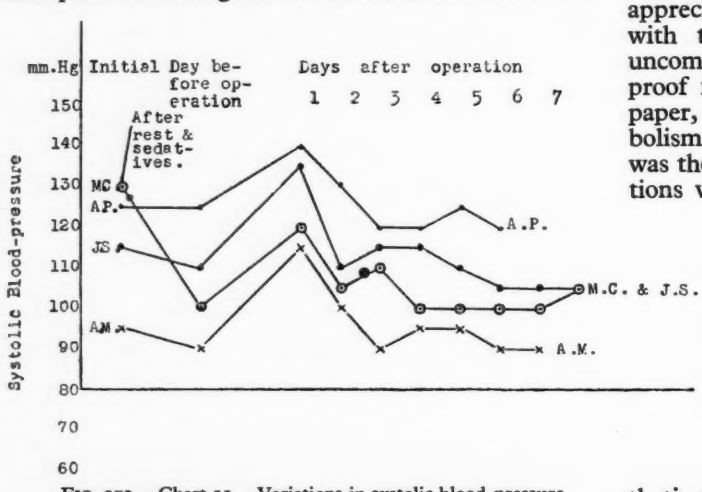


FIG. 272.—Chart 11. Variations in systolic blood-pressure after various types of operations. J. S., Subtotal thyroidectomy for non-toxic goitre; M. C., Subtotal thyroidectomy for thyrotoxicosis; A. P., For chronic appendicitis; A. M., For removal of secondary glands of neck.

DISCUSSION

After subtotal thyroidectomy for the relief of thyrotoxicosis, there is a post-operative reaction which is well-known to surgeons and nursing staff. This includes an increased pulse-rate, a raised metabolism, and fever, with the peak on the day after operation falling to a normal level in six to eight days. No satisfactory reason has been given to explain the fever, which is not an indication of infection. It is tempting to ascribe the whole post-operative reaction to a pouring out of thyroid secretion from the gland into the general circulation.

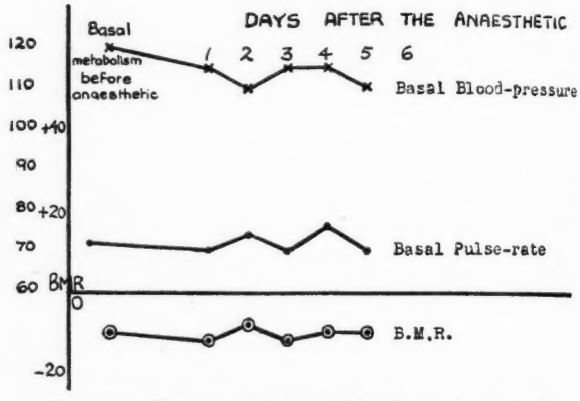


FIG. 273.—Chart 12. Showing effect of general anæsthetic on the basal metabolism.

There would appear to be support for this when it is appreciated that thyrotoxicosis is a disease associated with tachycardia, a raised metabolism, and not uncommonly a temperature above normal. Further proof for this concept seemed to be given in this paper, when it was found that the higher the metabolism on the day before operation, the more marked was the post-operative reaction. When the observations were extended, however, to include non-toxic goitre, tuberculous and malignant glands of the neck, and chronic appendicitis, it was found that the post-operative reaction was of the same degree as that obtained in thyrotoxicosis. It would appear, therefore, that the syndrome of tachycardia, increased metabolism, and fever following subtotal thyroidectomy for the relief of thyrotoxicosis is simply the result of a surgical operation and is not due to any specific thyroid factor.

In order to determine whether the anæsthetic was responsible for the post-operative reaction, a series of cases which were anæsthetized but not submitted to any surgical procedure involving trauma to skin vessels or organs were studied. It was found that the anæsthetic *per se* did not cause an increased basal metabolism or tachycardia.

SUMMARY AND CONCLUSIONS

1. After subtotal thyroidectomy for the relief of thyrotoxicosis there is a post-operative reaction which includes a raised basal metabolism, fever, tachycardia, and increased blood-pressure with

the peak on the day after the operation, and a return to normal in six to eight days.

2. This post-operative reaction is not specific for operations on a toxic goitre, but is present to the same degree after operations on non-toxic goitre, glands of neck, and for chronic appendicitis.

3. The syndrome is not present after the administration of an anæsthetic unaccompanied by any surgical trauma. It appears, therefore, that the typical reaction, which invariably follows an operation, is due solely to the effects of surgery and not to the anæsthetic.

III. THE EFFECTS OF IODINE MEDICATION AFTER SUBTOTAL THYROIDECTOMY FOR THYROTOXICOSIS

Plummer (1923) was the first to suggest giving iodine post-operatively in order to combat the severe reaction which invariably followed subtotal thyroidectomy for the relief of thyrotoxicosis. This was based on the assumption that the operative mortality was due to a lack of iodine, resulting in the secretion from the thyroid gland of an incompletely iodized product with very toxic properties. The administration of iodine after subtotal thyroidectomy according to Plummer caused a sharp fall in the post-operative deaths. In a later publication with Boothby (Plummer and Boothby, 1924) a careful study was made of the effects of iodine in thyrotoxicosis before operation. This paper, with others of a similar nature, has been discussed in the first section. In clinics where the teachings of Plummer and Boothby are observed, thyroid operations are carried out when iodine has brought about the maximum improvement, and this may vary from so short a time as six days to as long a time as twenty-five days. It is the custom in most clinics to continue the iodine after thyroid operations for several weeks or months. Thus Means (1937) gave iodine for ten days after operation, when the patient is discharged. Joll (1932) increased the iodine in twenty-four hours following operations, then reduced it to 10 min. of Lugol's solution daily for ten to twelve days. Among others, Pemberton (1926), Else (1929), and Jackson (1929) continued iodine for varying periods after operation. On the other hand, Davison and Aries (1939) believed that iodine administration after operation was not important. They compared a group of cases having iodine post-operatively with another group having no iodine, and they found no difference in the reactions as judged clinically and by the temperature, pulse-rate, and respiration.

The incidence of thyrotoxicosis following subtotal thyroidectomy for the relief of that condition has been well reviewed by Thompson, Morris, and Thompson (1930), and it ranges in different clinics from 0·25 to 25 per cent. Controversy still exists whether the thyrotoxicosis which occurs after subtotal thyroidectomy is due to a persistence or a recurrence of the disease. It has been suggested that if the basal metabolism remains within normal limits one to two months after iodine has been stopped, then any thyrotoxicosis subsequent to this is due to a recurrence of the disease, and any thyrotoxicosis previous to this is due to a persistence.

In the description of the cases which follows an attempt is made to determine the merits of giving iodine as a post-operative measure, and at the same time to find a means of assessing as early as possible a successful subtotal thyroidectomy.

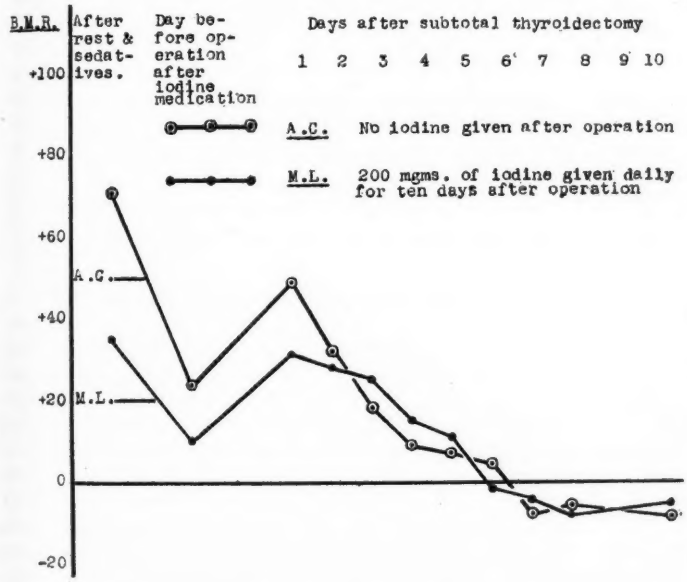


FIG. 274.—Chart 13. Showing the effect of iodine on the post-operative reaction following subtotal thyroidectomy for thyrotoxicosis.

EXPERIMENTAL

In order to observe the effects of iodine on the post-operative reaction following subtotal thyroidectomy for thyrotoxicosis, two groups of cases were studied. In one group iodine was commenced after the usual period of rest and it was continued for fourteen days after operation. In the second group iodine was stopped the day before operation. In Chart 13 (*Fig.* 274) typical curves of the basal metabolism in a case from each group are shown. It will be seen that the post-operative rises in metabolism on the day following operation are of the same degree, and in each case the basal metabolism falls to normal limits within a week. It is apparent, then, that, if patients are adequately prepared for operation with iodine, it is not necessary to continue its administration after the thyroid has been removed.

As iodine does not appear to play an essential part in the after-treatment of subtotal thyroidectomy, it was decided to investigate whether its administration had any disadvantages. It is well known that iodine causes the basal metabolism of thyrotoxicosis to fall, and that its action wears off after it has been stopped. The duration of the so-called iodine control of the basal metabolism and of the symptoms of thyrotoxicosis, both during its actual administration and after it has been stopped, is not well defined. An opportunity arose for making observations on the latter in a surgical case and the basal metabolism findings are plotted on Chart 14 (*Fig.* 275).

E. J., aged 18, was a case of thyrotoxicosis with the right lobe of the thyroid much more enlarged than the left, so that clinically she was labelled an adenoma of the thyroid. Iodine brought about a steady fall in the basal metabolism to the region of 3 in seventeen days, at which level the surgeon decided, instead of performing the customary subtotal thyroidectomy, to remove the right lobe of the gland only. Iodine was stopped on the day before operation, as a routine, and after an uninterrupted convalescence the basal metabolism was measured on the twelfth day after operation before discharge from hospital. The basal metabolism was + 20, i.e., higher than the level to which iodine had was then given for several days before as well as after operation. The beneficial effects of pre-operative iodine in thyrotoxicosis have been described in detail by Plummer and his colleagues at the Mayo Clinic. They showed that iodine brought about a marked clinical improvement and a fall in the basal metabolism, and that if operation was delayed until iodine had produced its maximum effect, the post-operative reaction was diminished accordingly.

It is the custom in most clinics to continue iodine for several weeks or months after thyroid operations. This appears to be due to two reasons. First, it is

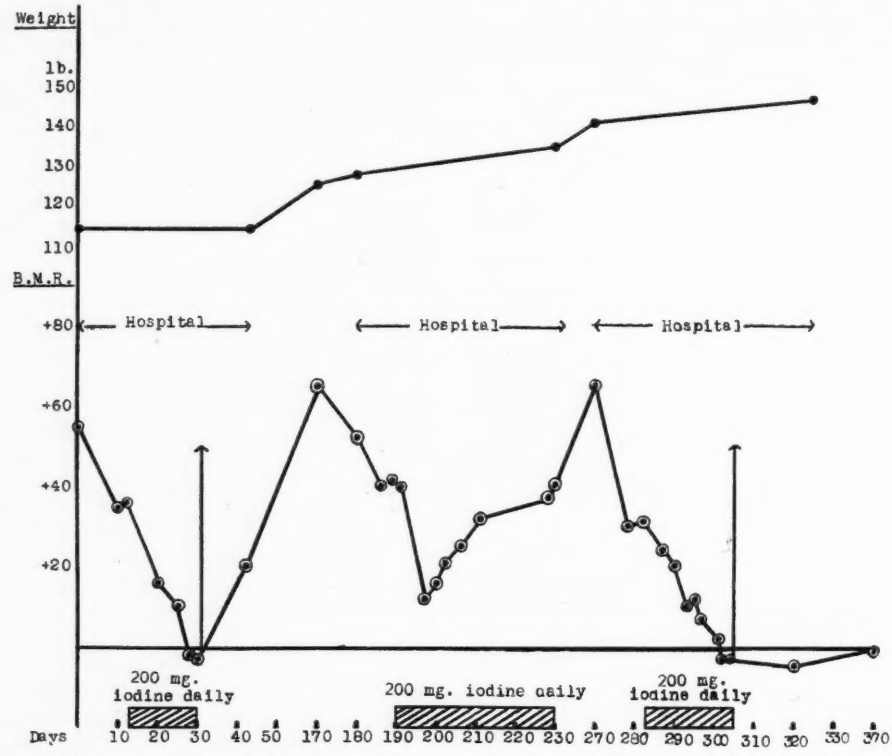


FIG. 275.—Chart 14. Basal metabolism findings in a study of iodine control in a surgical case of thyrotoxicosis.

reduced it before operation, and also raised above the limits of the normal controls. Despite the metabolism findings, a cure was claimed to have been established clinically. In four months' time the patient returned to hospital for a routine re-examination. By this time her basal metabolism was + 65, and clinically it was now obvious that thyrotoxicosis was present despite a gain of weight of almost 11 lb. She was taken into hospital for a further operation, and her progress is shown in Chart 14.

On iodine the basal metabolism fell from + 40 to + 12 in seven days, following which the patient became refractory to iodine and the basal metabolism began to rise despite iodine medication. Iodine was therefore stopped, but recommenced in six weeks' time. On this occasion a steady fall in the basal metabolism took place to the region of −3, at which level about seven-eighths of the gland remnant was removed. A satisfactory post-operative basal metabolism of −5 resulted.

DISCUSSION

Iodine was first given for several days *after* subtotal thyroidectomy for thyrotoxicosis in order to control the severe, often fatal, post-operative reaction. This procedure was later altered, and iodine believed that when the thyroid gland of a case of thyrotoxicosis is cut, and absorption of the gland toxin takes place into the general circulation, iodine is supposed to neutralize this toxin and control the resultant post-operative reaction. Secondly, it has been claimed that the post-operative administration of iodine prevents regrowth of thyroid tissue and a recurrence or a persistence of the thyrotoxicosis. With regard to the theory of a toxic absorption from the cut surfaces of the gland, evidence has been brought forward in a previous paper which suggests that this hypothesis is unlikely. Further, the administration of iodine after subtotal thyroidectomy did not control the increased basal metabolism in the days following the operation. The post-operative rise and decline in the basal metabolism was the same whether iodine was administered or not. Observations on the pulse-rate and body temperature agreed with the basal metabolism findings. It would appear, therefore, that iodine has no therapeutic value when given after subtotal thyroidectomy provided it has been given in adequate amounts before operation. The claim

that post-operative iodine can prevent regeneration of thyroid tissue and recurrence of the thyrotoxicosis is based on the observations of Marine and Lenhart (1909), who demonstrated that iodine inhibited compensatory hypertrophy of a normal thyroid remnant following removal of part of the gland. This view is not shared by the majority of workers, and it seems probable that iodine plays no part in the prevention of a persistence or a recurrence of the thyrotoxicosis after subtotal thyroidectomy. It is interesting to note that Thompson, Thompson, and Morris (1930) found little difference in the rate of recurrent thyrotoxicosis in the days before iodine was used (17·1 per cent) compared with the rate since the introduction of iodine (19·5 per cent). It appears, then, that recurrent or persistent thyrotoxicosis is independent of iodine administration, but rather it is related to some other cause such as the nature of the operation, the cause of the disease, or the like. As a corollary to the above, evidence has been brought forward in this paper to show that if iodine administration after subtotal thyroidectomy cannot do any good, then it probably may do harm. The administration of iodine after operation has the disadvantage of temporarily masking an active thyrotoxicosis, which will only become apparent after the iodine is stopped or until the patient becomes refractory to it. It is believed that if iodine is stopped on the day before subtotal thyroidectomy, then it will be possible to state by the twelfth day after operation (i.e., before the patient leaves hospital) whether a successful operation for thyrotoxicosis has been carried out or not. If the basal metabolism on the twelfth day after operation is significantly higher than the lowest pre-operative reading after iodine or above the limits of the normal controls, then the thyrotoxicosis is persisting and an inadequate amount of gland has been removed, and this despite absence of tachycardia, gain in weight, and other evidences of clinical improvement. In other words, the basal metabolism is a most sensitive index of the presence or absence of thyrotoxicosis.

SUMMARY AND CONCLUSIONS

1. If a case of thyrotoxicosis is given iodine in adequate amounts and subtotal thyroidectomy is carried out when the maximum clinical improvement with iodine has taken place, there is no need from a therapeutic point of view to continue iodine after the operation. Iodine given after subtotal thyroidectomy does not exert any control on the post-operative reaction. The post-operative rise and decline in basal metabolism, the pulse-rate, and body temperature in cases treated with iodine post-operatively were similar in those cases treated without iodine post-operatively.

2. If iodine is not continued after subtotal thyroidectomy it is possible to state by the twelfth day after operation whether the thyrotoxicosis has been relieved. If the basal metabolism on the twelfth day after operation is higher than the lowest pre-operative level or above normal limits, then the thyrotoxicosis has not been relieved. The basal metabolism is a more sensitive index of an unsuccessful operation for the relief of thyrotoxicosis than the pulse-rate, the weight, or the clinical symptoms.

The author has much pleasure in thanking the honorary staff of the Middlesex Hospital for permission to investigate their cases. He also has pleasure in acknowledging the gift of a metabolism machine from the Government Grants for Scientific Investigations, through the Royal Society.

REFERENCES

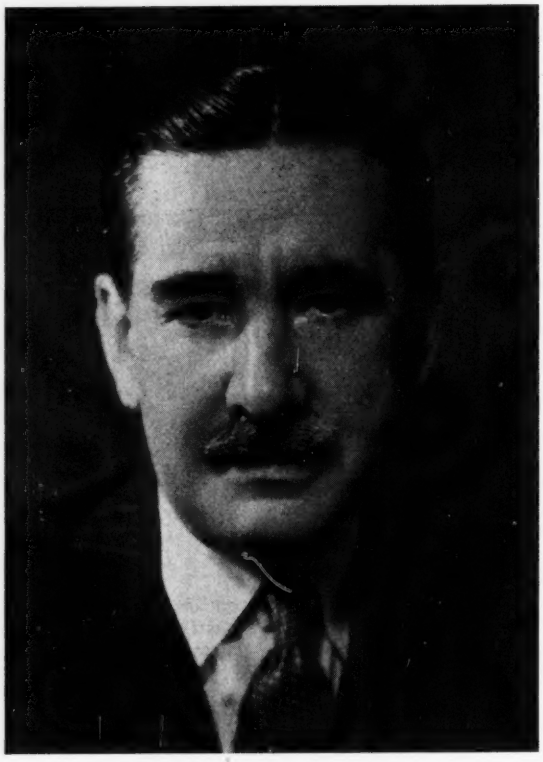
BARRETT, J. F., and ROBERTSON, J. D. (1937), *J. Path. Bact.*, **45**, 555.
BOOTHBY, W. M. (1921), *Coll. Pap. Mayo Clin.*, 469.
CHEADLE, W. B. (1869), *St. George's Hosp. Rep.*, **4**, 175.
COINDET, J. R. (1820), *Ann. Chim. Phys.*, **15**, 49.
COURTOIS, M. B. (1813), *Ann. Chim. Phys.*, **88**, 310.
COWELL, S. J., and MELLANBY, J. E. (1924), *Quart. J. Med.*, **18**, 1.
DAVISON, M., and ARIES, L. J. (1939), *West. J. Surg.*, **47**, 589.
DUBOIS, E. F. (1936), *Basal Metabolism in Health and Disease*, 3rd ed., 342. London: Baillière, Tindall & Cox.
EBERTS, E. M. (1921), *Canad. med. Ass. J.*, **11**, 641.
ELSE, J. E. (1929), *Endocrinology*, **13**, 40.
FITZGERALD, R. R. (1926), *Canad. med. Ass. J.*, **16**, N.S., 159.
FULTON, M. N., and AULT, H. L. (1930), *New Engl. J. Med.*, **203**, 327.
JACKSON, A. (1929), *Amer. J. Surg.*, **6**, 7.
JAGIČ, N., and SPENGLER, G. (1924), *Wien. klin. Wschr.*, **37**, 116.
JOLL, C. A. (1932), *Diseases of the Thyroid Gland*. London: Heinemann.
KOCHER, T. (1910, a), *Arch. klin. Chir.*, **92**, 1166.
— — (1910, b), *Brit. med. J.*, **2**, 1931.
LERMAN, J. (1930), *New Engl. J. Med.*, **203**, 254.
— — and MEANS, J. H. (1931), *Amer. J. med. Sci.*, **181**, 745.
LOEWY, A., and ZONDEK, H. (1921), *Dtsch. med. Wschr.*, **47**, 1387.
LUGOL, J. G. A. (1830), *Memoir on the Employment of Iodiuretted Baths in Scrofulous Diseases, followed by a Table for their Administration according to Age*, 34. Paris.
MARINE, D., and LENHART, C. H. (1909), *Arch. int. Med.*, **4**, 440.
— — — — (1911), *Ibid.*, **7**, 506.
MARKWICK, A. (1846), *Medical Times*, **14**, 446.
MEANS, J. H. (1937), *Thyroid and Its Diseases*. Lippincott: Philadelphia.
NEISSER, E. (1920), *Berl. klin. Wschr.*, **57**, 461.
OHLEMAN, M., (1911), *Ibid.*, **48**, 385.
PEMBERTON, J. DE J. (1926), *Coll. Pap. Mayo Clin.*, **18**, 550.
PLUMMER, H. S. (1923), *J. Amer. med. Ass.*, **80**, 1955.
— — and BOOTHBY, W. M. (1924), *J. Iowa St. med. Soc.*, **14**, 66.
PROUT, W. (1834), *Chemistry, Meteorology, and the Function of Digestion Considered with Reference to Natural Theology*, 113. London: William Pickering.
ROBERTSON, J. D. (1937), *Lancet*, **2**, 815.
— — (1944), *Brit. med. J.*, **1**, 617.
SEGALL, H. N., and MEANS, J. H. (1924), *Arch. Surg.*, **8**, 176.
STARR, P., WALCOTT, H. P., SEGALL, H. S., and MEANS, J. H. (1924), *Arch. int. Med.*, **34**, 355.
STURGIS, C. C. (1923), *Ibid.*, **32**, 50.
THOMPSON, W. O., BAILEY, A. G., THOMPSON, P. K., and THORP, E. G. (1930), *Ibid.*, **45**, 261.
— — and MORRIS, A. E. (1930), *West. J. Surg.*, **38**, 763.
— — — — and THOMPSON, P. K. (1930), *Ibid.*, **46**, 946.
— — and THOMPSON, P. K. (1931), *Ibid.*, **48**, 351.
TROUSSEAU, A. (1868), *Lectures on Clinical Medicine*, **1**, 587. Lecture 19. The New Sydenham Society, London.
WALLER, H. E. (1914), *Prescriber*, **8**, 153.

In Memoriam

ARTHUR TUDOR EDWARDS

(1890—1946)

THE death of Tudor Edwards has robbed the world of a master-surgeon and Britain of the most brilliant operator in the domain of thoracic surgery. Though only 56 years of age, he had long acquired a veritably international reputation, and his pupils, assistants,



TUDOR EDWARDS
(1890—1946)

and those who had come under his influence and spell are to be numbered in every Dominion and in many countries outside the British Empire.

The years of early training were spent in Middlesex Hospital, a fruitful nursery of surgical operators. Tudor Edwards served in two wars; doubtless his experience of forward surgery in the 1914–18 armageddon first directed his thoughts towards the thorax as his own future surgical province; the recent world war found him giving of his knowledge and experience in the role of consultant for injuries and diseases of the chest to the Ministry of Health, to the War Office, and to the Royal Air Force. Tudor had rightly approached the specialty which was to make him world-famous along the avenue of general surgery, and his contacts with such men as Bland-Sutton, Moynihan, and Cuthbert Wallace cannot have failed to exert some influence on his surgical outlook.

His chest surgery was learned from no other pioneer, but carved out of the hard rock of personal experience. He was perhaps fortunate in the exact moment of his surgical activity, for the great advances in anæsthesia facilitated his work, and Tudor Edwards was also blessed in the co-operation of wise physician-colleagues, particularly at Brompton Hospital, who were possessed of vision and who reposed their complete confidence in him. To-day outstanding surgical achievements are not consummated by the lone worker, but by the man who can organize and inspire colleagues, assistants, and nursing staff to form one harmonious loyal unit. The leadership of Tudor Edwards attracted around him such a coterie of acolytes. The number of hospitals he served was legion: Westminster, the London, Queen Mary's, Roehampton, Midhurst, Millbank, and many others; doubtless his heart lay in Brompton, but he served each institution with fidelity and brought lustre and distinction wherever he was attached.

Surgical honours came to him in abundance from colleagues at home and from universities and learned associations abroad; he was an Honorary M.D. of Grenoble and of Oslo Universities; an Honorary Fellow of the American Society of Thoracic Surgeons; he had occupied the presidential chair of various British associations concerned with the chest; he was a valued member of the Council of the Royal College of Surgeons of England. The written word is often a meretricious avenue to international reputation; Tudor Edwards's fame has rested and will rest on his deeds rather than words, although 33 papers under his name are chronicled in the *Cumulative Index* and there was also that last notable paper in the first number of the new journal *Thorax*, in which he summarized his experiences in over 1000 cases of bronchial carcinoma, including 70 cases in which he had performed pneumonectomy or lobectomy for this disease.

Despite the menace of a malady which finally carried him off, he continued to soar aloft to the surgical empyrean, disdaining the black cloud of death which threatened his life so long. The amount of work which he accomplished was prodigious and more and more exacting in its character. Like the Emperors Vespasian and Hadrian, who when reproved for activities prejudicial to failing health replied, "An Emperor must die standing", Tudor obstinately refused to spare himself. Perhaps no man has borne so heavy a medical burden, for there were few physicians or surgeons who did not turn to him for help for themselves or their kith and kin when stricken with thoracic maladies.

The man who achieved all this was no leaden superman, but a vital figure with all the charms and graces. The pioneer is seldom the exploiter, but Tudor Edwards was happily spared to see the water of early conviction become the red wine of

established faith. He lived to enjoy the satisfaction, not only of gazing retrospectively on his own brilliant pioneer achievements, but of looking forwards into the promising vista which his work had opened up. Well might he have exclaimed in the words of Valiant-for-Truth, "Yet now I do not repent me of all the trouble I have been at to arrive where I am. My sword I give to him that shall succeed me in my Pilgrimage, and my Courage and Skill to him that can get it. . . ."

*Pulchrumque mori succurrit in armis.**

* *Aeneid*, ii, 317.

SHORT NOTES OF RARE OR OBSCURE CASES

A CASE OF ACUTE PANCREATITIS WITH SUBCUTANEOUS FAT NECROSIS

By HUGH BLAUVELT

SURGEON, NORTH MIDDLESEX COUNTY HOSPITAL, LONDON

FAT necrosis due to acute pancreatitis is commonly seen within the abdomen where it affects the mesentery, the omentum, the pancreas, and the peritoneum. Outside the abdominal cavity fat necrosis is rare except in the extraperitoneal fat. The occurrence of necrosis in more distant regions and structures —the mediastinum, the pleura, the pericardium, the marrow of long bones, and the peri-articular fat— has been observed, but except in connexion with the experimentally produced acute pancreatitis of animals no record can be discovered of necrosis developing in the subcutaneous fat.

In the case of acute pancreatitis herein described areas of fat necrosis were present in the subcutaneous fat covering most of the body.

CASE REPORT

M. C., a married woman 41 years old, was admitted to the North Middlesex County Hospital on Oct. 27, 1939, complaining of epigastric pain of two days' duration. Nausea and vomiting occurred the day before she entered hospital. The bowels were constipated and micturition was difficult. The previous history revealed no detail of importance.

ON ADMISSION.—The patient looked seriously ill. T. 99·6°, P. 120, R. 24. The abdomen was not distended, but there was generalized tenderness and rigidity and

FIG. 276.—Showing the pigmented areas in the right thigh.

these were more marked over the gall-bladder. The diagnosis lay between acute cholecystitis and acute pancreatitis.

The following day many red areas resembling the eruptions of erythema nodosum appeared upon the skin of the legs and arms.

FIRST OPERATION.—On Oct. 29 laparotomy was decided upon. The incision in the abdominal wall revealed necrosis of the extraperitoneal fat and the peritoneal cavity was not opened. A portion of one of the superficial lesions on the right leg was excised and the cut surface showed the typical appearance of fat necrosis.

DESCRIPTION OF THE SKIN LESIONS.—The superficial areas of fat necrosis were confined to the arms, legs, and buttocks, with the exception of two lesions which were

situated on the back immediately to the right of the 11th dorsal spine. All the lesions appeared during the first week of the illness, but two were discovered for the first time five weeks after her admission to hospital. Of these two nodules, one was about 2 in. below and to the left of the umbilicus, and the other was situated in the subcutaneous tissue of the right flank.

The distribution of the lesions in the affected parts was not uniform, the most extensively involved areas being the upper and outer aspects of the thigh and the tissues close to and below the knee. The lesions on the right leg were more numerous than those on the left.

In size the lesions varied from a mark no larger than a pin-prick to a round or oval patch 2 in. or more in diameter; the majority measured about ½ in. across. All the lesions exhibited the same round or oval shape and in no case was any marked irregularity in the outline observed. (*Fig.* 276.)

The surface of several of the spots was slightly raised above the level of the surrounding skin. Unlike the remaining and more numerous ones, these plaques were softer in the centre than towards the periphery and they thus resembled small abscesses; suppuration, however, did not occur in any one of them.

Microscopical examination of the portion of tissue removed at operation displayed the characteristic features of fat necrosis and showed no evidence of cellular activity or of vascular reaction.

TESTS AND INVESTIGATIONS.—

Urinalysis (Dec. 21): Catheter specimen of urine—red blood cells, pus, and *B. coli*. These abnormalities no doubt were due to infection from previous catheterization.

Urinary Diastase: Oct. 30, 2400 units for 24 hours, representing an index of 10; Dec. 21, 8700, or an index of 6.

Blood-sugar (Nov. 3): 90 mg. per 100 c.c.

W.B.C.: Oct. 30, 12,500; Dec. 21, 7200.

Loewi's mydriatic reaction, positive.

Radiographs of the abdomen showed signs of gall-stones. Radiographs of the limbs on several occasions showed nothing abnormal until July, 1940, when vague shadows were present suggestive of calcification in the necrotic lesions.

CLINICAL PROGRESS.—Retention of urine occurred during the first six days following operation and was treated by repeated catheterization. Eight days after operation 10 oz. of fluid containing yellow flakes was vomited. Unfortunately this was thrown away, but the yellow substance was described by a nurse as being "like soft butter". Up to this date in hospital the patient had received no fat in her diet.

Recovery proceeded slowly: she left hospital on Dec. 21, about eight weeks after admission.

When examined as an out-patient seven months after the beginning of the illness all external signs of fat necrosis had disappeared with the exception of a few rose-coloured areas on the thighs. No abnormal thickening was detected in any of these patches.

SECOND OPERATION.—On Aug. 7, 1940, she was re-admitted to hospital and cholecystectomy was performed on the following day. The operation presented no difficulty and two small stones and a chronically inflamed gall-bladder were removed. On Aug. 10 she became comatose and death followed 24 hours later. The blood-urea before death was 200 mg. per 100 c.c.

POST-MORTEM EXAMINATION.—The abdomen showed no sign of infection, hæmorrhage, or bile leakage. The liver, bile-ducts, and pancreas were normal and no evidence was found of recent or of old fat necrosis. The kidneys were small, contracted, and granular, and no doubt death was due to chronic nephritis. In the limbs no calcified areas were found.

COMMENT

Fat necrosis in acute pancreatitis is due to the action of lipase upon the neutral fat of the tissues. The immediate result of this reaction is the production of glycerin and fatty acid. Following this the fatty acid unites with calcium to form an insoluble soap. This soap is the essential constituent of the opaque plaques which characterize fat necrosis.

The Transportation of Lipase to the Site of the Lesion.—Lipase may be carried from the diseased pancreas to the various regions where fat necrosis has been observed in a number of ways. The lesions so frequently found in the peritoneum and mesentery and in the pancreas itself are usually regarded as the result of the direct spread of the liberated ferment. When the extraperitoneal fat, the pleura, the pericardium, and the mediastinum are affected the lipase is said to travel by way of the lymphatics. Necrotic lesions in the bone-marrow and peri-articular tissues have been attributed to pancreatic emboli travelling through the blood-stream. In the case of subcutaneous fat necrosis described in this paper such a method of dissemination would best account for the occurrence of the lesions.

A study of the blood-vessels draining the pancreas suggests that when the gland is acutely inflamed favourable conditions exist for the transference of minute particles of pancreatic tissue to the portal blood-stream. The portal vein is formed near the head of the pancreas by the union of the splenic and superior mesenteric veins. The pancreas itself is drained by many small vessels which open directly into the splenic, the superior and inferior mesenteric, and the veins along the greater curvature of the stomach. When the head of the pancreas is inflamed and swollen the portal vein is likely to be pressed upon close to its origin. As a result of this venous obstruction the pancreas becomes congested and microscopical portions of tissue may be absorbed and eventually reach the systemic circulation.

Involvement of Stomach and Bladder.—In the author's case the possibility of submucous lesions in the stomach and their separation and appearance in the vomited material cannot be overlooked. The fact that no blood was observed in the stomach contents is evidence, but not proof, against such an occurrence.

Dysuria is not a rare symptom in acute pancreatitis and it may be explained by the presence of areas of fat necrosis underneath the peritoneum of the bladder. A deposit of this nature in contact with the muscle tissue of the bladder might interfere with the neuromuscular mechanism of micturition.

A CASE OF CONGENITAL MULTIPLE ARTERIOVENOUS FISTULÆ OF THE HAND

By J. A. WHITE

SURGEON, PARK PREWETT HOSPITAL, BASINGSTOKE

CONGENITAL arteriovenous fistula of the extremities would appear from the literature to be a rare condition. Pemberton and Saint (1928) reported 9 cases at the Mayo Clinic between 1916 and 1928. Lewis (1930) collected 24 cases from the literature and described 6 of his own. Other writers have reported one or two cases. Horton (1932), however, produced a series of 23 from the Mayo Clinic between 1929 and 1931. Some of these were only diagnosed by the venous oxygen content, and had no murmur or thrill, so it is possible that the condition is not so rare, but sometimes escapes recognition. Bone changes and local gigantism are reported in many of the cases, but malacia of the semilunar has not been mentioned.

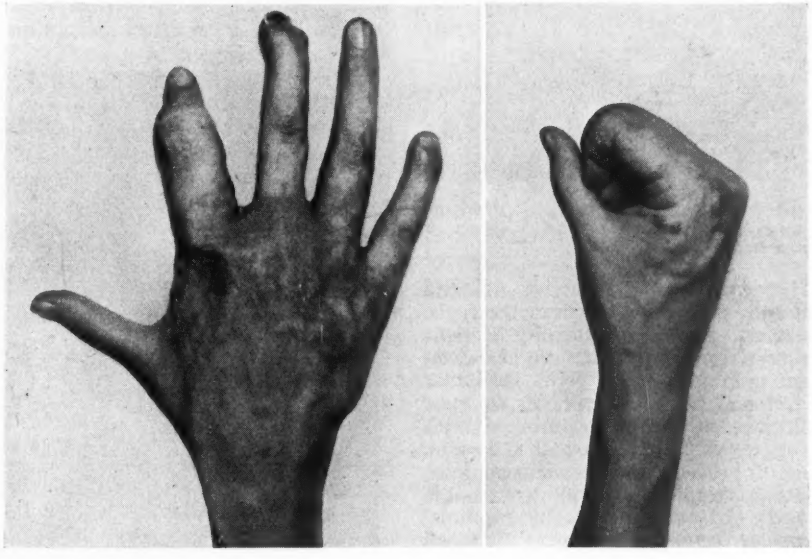


FIG. 277.—Photographs of affected right hand, showing deformities, dilated veins, and normal thumb.

CASE REPORT

A girl aged 12 was first seen on Feb. 7, 1943. The mother stated that a purplish discoloration of the right hand had been present from birth. Disproportionate increase in the size of the hand, associated with some deformity, had been noticed in the last two or three years. The patient's only complaint was of mild pain in the deformed fingers and in the wrist.

ON EXAMINATION.—The patient was an intelligent healthy girl. The right hand showed a large irregular port-wine stain on both palmar and dorsal surfaces. The whole hand, with the exception of the thumb, which was quite normal, was swollen and covered with dilated tortuous veins. Dilated veins were also present in the forearm. The four fingers showed gigantism and deformities (*Fig.* 277). A thrill was palpable, maximum in the palm. In addition to the radial and ulnar arteries, four deep pulsating vessels could be palpated, one on the palmar surface and three on the dorsal surface. The hand was used by the patient for all ordinary purposes, and its function was fair. Elevating the arm caused the dilated veins to disappear. With the stethoscope a continuous-cycle bruit with systolic accentuation could be heard over most of the hand, but was maximum on the palmar surface at the base of the first finger. The bruit was high-pitched and musical, and could be heard conducted as high as the right supraclavicular fossa. No murmur could be detected above the wrist when a tourniquet had first been applied at this level, thus proving the arteriovenous communications responsible for murmurs to be at or below the wrist. This was not absolute proof, however, that other communications did not exist higher up, as cases have been described without murmurs (Horton, 1932).

The right hand was definitely warmer, and usually moister, than the left. The heart showed no clinical signs of enlargement or abnormality. The right humerus, radius, and ulna were approximately half an inch longer than the left.

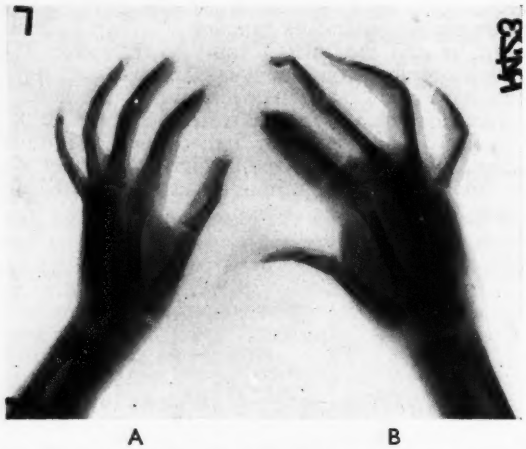
The blood-pressure findings were interesting and were taken with the experimental model of the M.E.M. sphygmoscope (Mendelssohn et al., 1945) as it was found to be very difficult to determine the pressure in the

SITE	SYSTOLIC PRESSURE	DIASTOLIC PRESSURE
	(mm. Hg)	(mm. Hg)
Affected upper arm (R.)	125	40
Normal upper arm (L.)	95	60
Normal upper arm (L.) (with compression of R. brachial artery)	95	50
Right thigh	120	50

affected arm with the ordinary blood-pressure machine. This instrument has since been found very helpful in taking distal limb pressures in traumatic aneurysms.

At rest the pulse-rate was 90, and it dropped to 83 with compression of the right brachial artery (Branham's or Nicoladoni's sign).

X-ray Findings (*Figs.* 278, 279).—Report by Dr. Feuchtwanger :—



A B

FIG. 278.—A, Radiograph of affected hand; B, Radiograph of normal hand for comparison.

"*Both Hands*: Hyperostosis, i.e., excessive size and development, of right 2nd, 3rd, 4th, and 5th metacarpals. Thumb of normal size and length. Deformity of right radial and ulnar epiphysial regions, with slight lateral displacement of radial epiphysis. The right semilunar is smaller than the left and shows irregularity of bone structure, i.e., localized bone atrophy combined with beginning sclerosis, appearances of Kienböck's disease. Extensive destruction of proximal interphalangeal joint of index finger and distal interphalangeal joint of middle finger. Shafts of 2nd, 3rd, and 4th right proximal phalanges show irregular trabeculation and increased bone striation as seen usually in hæmangioma. The left hand shows no radiological abnormality. *Arteriogram*: Injection into right radial artery about 1½ in. above wrist-joint. Right radial artery filled. No filling of thumb area. Extensive arterial filling of palmar arch with marked accumulation of opaque medium between heads of 2nd and 3rd metacarpals. Soft tissues of index finger show vascular filling of moderate density. Venous dilatation in lateral carpometacarpal region."

The arteriogram suggests multiple fistulæ but gives no definite information as to number or site. It also suggests small aneurysmal sacs in the region of the 2nd and 3rd metacarpophalangeal joints, and this was the region which was first attacked surgically. Another arteriogram into the mid-brachial artery, but not giving such a good picture, is not included. It gives no further information, but it also fails to show filling of arteries to the thumb.

TREATMENT AND PROGRESS.—Injections of sclerosing fluid (5 per cent sodium morrhuate) into regional veins and subcutaneous injections over the site of maximum murmur had no effect. Incidentally, the maximum murmur has recently, in some traumatic arteriovenous fistulæ, been found at some distance from the actual lesion. This has been on the proximal side of the fistula, where a deep vein, in its course back to the heart, has been approaching the surface.

Four attempts at surgical extirpation over a period of eight months produced no clinical improvement, and the patient was discharged.

Follow-up discovered that a severe hæmorrhage had later necessitated amputation through the forearm at another hospital.

DISCUSSION

Treatment.—Surgery and sclerosing injections failed to produce any improvement. The condition can only be cured by closing all the fistulæ, and in most cases they are multiple and minute. Surgery is also made very difficult by the angiomatous tissue. Horton (1932) states that "apparently" only three cases have been cured by surgical measures. He advocates injection of the regional veins with a sclerosing fluid in an attempt to obliterate the abnormal communications, and reports that in one case there was an "apparent" cure. In most cases it is probably best to do nothing until the heart is affected, and then amputation will be necessary. It is possible that the unsuccessful surgery in this reported case precipitated the necessity for amputation before the heart was showing any signs of being affected.

FIG. 279.—Arteriogram of vessels of right hand. Injection was into radial artery just above wrist.

Bone Changes.—This case shows hypertrophy, rarefaction, epiphysial changes, joint destruction, and a patchy sclerosis of the semilunar. All the changes must be attributed to the vascular disorder, but it is interesting to speculate on the mechanism. The hypertrophy and epiphysial changes must surely be due to the increased blood-supply to the limb, and occurred in the radius, ulna, and humerus, which were probably above the fistulæ, as well as in the hand. The rarefaction and joint destruction are certainly due to invasion by the angiomatous

tissue. It is difficult to explain the complete normality of the thumb, except by postulating a separate blood-supply from high in the upper arm, which seems improbable, although arteriograms with injection into the brachial artery in the middle of the upper arm and the radial just above the wrist failed to show any of the arteries to the thumb.

The generally accepted view of Kienböck's disease is that it is fundamentally due to a disturbance of blood-supply. Many authors have suggested injury as the cause of this disturbance, but it is interesting to note that Cave (1939) could only find one case in the literature following palmar dislocation, where the main posterior blood-supply must be damaged. Sclerosis often occurs, but revascularization and not collapse follows. Jackson Burrows (1941) stresses the differences between aseptic necrosis and osteochondritis, and produces some experimental evidence that cutting off the arterial blood-supply produces aseptic necrosis and not osteochondritis. He suggests that either venous obstruction or hæmatoma formation within the nucleus may be the causal factor of osteochondritis. In this case one can imagine an arteriovenous communication between the main nutrient artery and vein just proximal to the semilunar. This would cut down the arterial supply to the bone, but would also increase the pressure in the vein with consequent venous obstruction. Other suggestions have been reflex vasomotor constriction (Leriche), embolic occlusion (Axhausen), endarteritis obliterans and thrombosis (Witts), and Mouatt et al. have described the conditions following phlegmon of the hand and scarlet fever. The above authors were quoted by Roth (1943).

Blood-pressure Findings.—The interesting fact here is the great increase in systolic pressure in the affected arm. The possibility that this might be due to a temporary rise of general pressure following occlusion of the arteriovenous communications while taking the reading, was ruled out by taking the pressure in the normal arm while occluding the blood-supply to the affected side.

The systolic pressure was unaltered and the diastolic slightly lowered, as shown in the table of blood-pressures. Holman (1937), in his experimental fistulæ, reports considerable rise in general blood-pressure, both systolic and diastolic, when the fistula is occluded. That this did not happen in this case can be explained partly by the minuteness of the communications (the small drop in pulse-rate is additional evidence of this), and partly by the adaptability of a youthful circulation.

Holman also describes the enlargement of proximal vessels which we know occurs above a fistula. The blood-pressure in a normal leg is higher than that in a normal arm, and larger vessels are mainly the cause of this. The increased systolic pressure in the affected arm in this case may likewise be attributed to the larger vessels.

The lower diastolic pressure in the affected arm can be explained by the diminished peripheral resistance due to leakage through the fistulæ.

Horton (1932) records the pressures of both limbs in four of his cases. In three the pressure was much higher on the affected side, and in one a little lower.

REFERENCES

CAVE, E. F. (1939), *J. Bone Jt. Surg.*, Oct., 858.
HOLMAN, E. (1937), *Arterio-venous Aneurysm.* Macmillan.
HORTON, BAYARD T. (1932), *J. Amer. med. Ass.*, **98**, 373.
BURROWS, H. JACKSON (1941), *Brit. J. Surg.*, **29**, 23.
LEWIS, D. (1930), *Lancet*, **2**, 621, 680.
MENDELSSOHN et al. (1945), *Brit. J. Anæsth.*, **19**, 120.
PEMBERTON, J. de J., and SAINT, J. H. (1928), *Surg. Gynec. Obstet.*, **46**, 470.
ROTH, F. B. (1943), *J. Bone Jt. Surg.*, July, 683.

A CASE OF MELORHEOSTOSIS

BY A. DAVID LE VAY, LONDON

THE purpose of this paper is not to supply an exhaustive survey of melorheostosis, but to add a fourth case to the three cases hitherto published in the British literature.

Boggon (1938) described a case in a man of 30, in whom the right side of the pelvis, the right tibia and fibula, and the bones of the right foot were affected, in association with gummatous ulceration.

Fairbank (1939) regarded a case of Wakeley's, a boy of 8 in whom both legs were affected, as a true instance of melorheostosis and a link with the generalized form of osteosclerosis (Albers-Schönberg). This is incidentally the only case ever described with well-marked bilateral features.

Franklin and Matheson (1942) recorded a very extensive involvement of the skull, spine, pelvis, and the bones of the arm and leg, all on the right side, in a woman of 41.

The case described by Hall (1943), and that of Hilton (1934) quoted by Hall, cannot be regarded as true instances of the disease, for the X-ray appearances are quite atypical.

The present findings were an accidental radiographic discovery in a boy of 9 with mild knock-knee. There is involvement of the left ischium, the outer half of the lower femoral epiphysis, both fibular epiphyses, the talus, cuneiforms, and third and fourth metatarsals and phalanges, all on the left side (*Figs.* 280–283). There were no symptoms, and no signs apart from a palpable thickening on the dorsum of the foot. Serum-calcium, plasma-phosphorus, and phosphatase were normal.

The purely epiphysial location in the fibula and femur is interesting and somewhat unusual. It will be noted that the shaft of the fibula, though not sclerosed, is almost twice the thickness of the right, with considerable blurring, and is presumably exhibiting presclerotic changes. There are similar changes in the upper half of the left femur.

FIG. 280.—Sclerosis in the left ischium; thickening of the shaft of the left femur.

FIG. 281.—Sclerosis in the outer half of the left lower femoral epiphysis and in the upper epiphysis of the fibula.

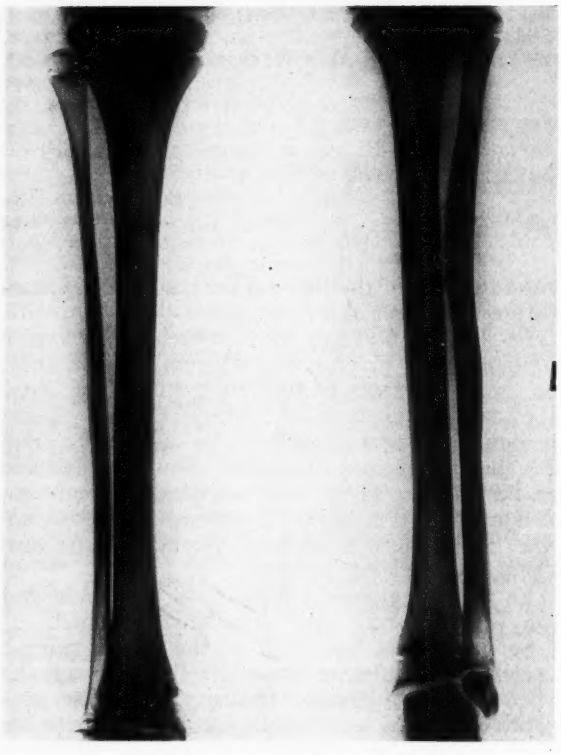


FIG. 282.—Deposits in both left fibular epiphyses, with blurring and thickening of the fibular shaft.

FIG. 283.—Splashes of sclerosis in the cuneiforms, metatarsals, and phalanges of the left foot.

REFERENCES

BOGGON, R. H. (1938), *Proc. R. Soc. Med.*, **32**, 439.
FAIRBANK, H. A. T. (1939), *Brit. J. Surg.*, **27**, 1.
FRANKLIN, E. L., and MATHESON, I. (1942), *Brit. J. Radiol.*, **15**, 185.
HALL, G. S. (1943), *Quart. J. Med.*, **12**, 77.
HILTON, G. (1934), *Lancet*, **1**, 122.

CONSTRICTION OF THE EXTENSOR POLLICIS BREVIS TENDON

AN UNUSUAL LESION SIMULATING DE QUERVAIN'S DISEASE

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SURGEON LIEUTENANT COMMANDER, R.N.V.R.

STENOSING tenovaginitis (de Quervain's disease) is a well-recognized and fully documented condition, first described in 1895 (Scheidner, 1928). In adults it usually affects the extensor pollicis brevis or the abductor pollicis longus, and in children has been described (Zadek, 1942) involving the flexor pollicis longus. In the common extensor type the patient complains of pain in the region of the radial

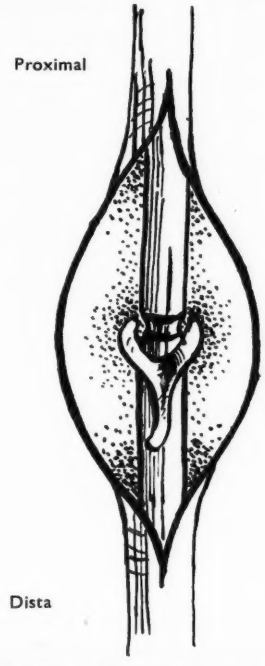

FIG. 284.—Diagram of appearance on opening tendon-sheath. The long limb of the occluding structure detached from the roof of the sheath; the two short limbs attached to floor of sheath and the whole embracing the tendon tightly but unattached to tendon. Note the groove in the tendon from which the structure slipped off with a jerk.

styloid and of difficulty in straightening the thumb, such that manipulation may be required. Passive movements meet with resistance and the thumb may extend with a definite 'snap'. Classically, adduction at the wrist is free if the thumb be free, but is limited and painful if the thumb is included with the fingers in testing this wrist movement. There may be slight swelling over the affected tendon. The pathology tends to occur where the abductor pollicis longus and the extensor pollicis brevis pass beyond the osteofibrous canal overlying the lower end of the radius, and on exposure there is seen a fibrous thickening of the tendon sheath. This tissue is hard and cuts like gristle, and it restricts the free play of an otherwise normal tendon. The histology suggests the reaction of trauma rather than infection, and certainly many cases occur in women using their hands with, for example, the monotonous regularity of housework. Treatment may be conservative or surgical. Immobilization may reduce the swelling of the sheath in an early stage of the condition. More satisfactory to surgeon and patient alike is the operative exposure of the tendon under local anæsthesia; the sheath is laid open and the free action of the thumb can at once be demonstrated.

Such was the condition in mind in examining and submitting to operation the patient described below: but an unusual and unexpected lesion was disclosed.

CASE REPORT

A naval officer aged 42 was admitted to hospital on July 18, 1945, complaining of pain in the right thumb. He recorded seven months' disability due to pain at the base of the thumb. There was no history of trauma, but he ascribed the condition to excessive writing during the previous year. Full abduction was difficult because he "felt something catching" and at times he noticed a "click" as he extended the thumb.

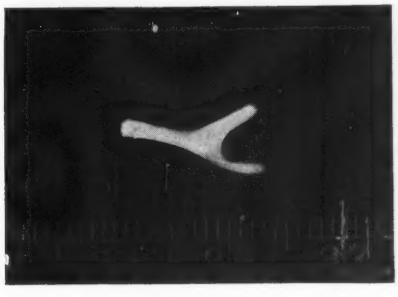

FIG. 285.—Photograph of structure as it appeared after excision. View of under-surface. (× ⅔.)

ON EXAMINATION.—He was in perfect health. He pointed to the line of the tendon of extensor pollicis longus as the site of a radiating pain from wrist-joint level to the interphalangeal joint of the thumb. On palpation there was tenderness just distal to the radial styloid in the line of the extensor pollicis brevis. All movements were quite free, but there was slight pain on full extension of the thumb referred to the tender area. He considered that the symptoms were variable and that at the time of examination the condition was somewhat less troublesome. No definite click was heard on movement. When examined on a previous occasion symptoms had been more marked and the adduction test noted above was positive. Although there was no palpable swelling, a pre-operative diagnosis of de Quervain's disease was made. At his own request a general anæsthetic was arranged.

OPERATION (July 19).—With a pneumatic tourniquet on the upper arm an incision was made along the line of the extensor pollicis brevis from the radial styloid 1 in. distally. A tendon-sheath of normal thickness and consistency was exposed and slit open in the length of the incision, to reveal the tendon tethered by a peculiar band of tissue (*Fig.* 284). The paired limbs of this structure arose from the floor of the sheath on each side of the tendon and embraced the latter tightly over the volar

aspect—tightly enough to cause a local constriction of the tendon. The single limb merged with the roof of the sheath and reference to *Fig.* 285 indicates the size of the structure. On extension of the thumb the band jumped out of the groove it had printed on the tendon—the intermittent symptoms were thus readily explained. The obstruction was excised by dividing it at the three attachments to the sheath. A probe was passed distally for an inch and no further obstruction was encountered. Extensor pollicis brevis may share a common canal with abductor pollicis longus at this point, but in this case the tendons had separate compartments. It was considered wise to inspect the adjacent sheath, but on slitting it open no abnormality was found. The abductor sheath was approximated with two fine catgut sutures: the abnormal sheath was left open. The skin was closed with silk sutures.

POST-OPERATIVE COURSE.—Full movements were allowed two days after operation and were painless and free. The stitches were removed on the tenth day with primary healing. On Aug. 18 the doctor who had originally submitted the patient wrote to say that the latter was completely satisfied with the result of operation and free from all symptoms.

MICROSCOPICAL FINDINGS.—Microscopical examination of a cross-section of a Y-shaped fibrocartilaginous structure showed a reticulum of collagen fibres with cells interspersed and resembling normal tendon cells. The outer cells were somewhat flattened, but there was no true capsule or sheath. There was no evidence of inflammation and the appearance was of normal tendon.

DISCUSSION

The interest in this case lies in the differential diagnosis. Not surprisingly, it was mistaken for stenosing tenovaginitis. The aetiology is uncertain, but the symmetrical appearance and the absence of thickening or adhesions in the adjacent sheath are against an inflammatory origin. The histology suggests a normal connective-tissue structure. The arrangement was not akin to that of the vinculæ of tendons which carry vascular bundles and which occur more distally in the fingers and toes. A congenital origin is more likely and no reference to such has been traced in the literature available for study. The delayed onset of symptoms may have been due to a reactionary thickening of the structure induced by the unusual amount of writing done by the patient in recent months.

Treatment is surgical, with identification and removal of the constriction. Forcible manipulation might have snapped this structure and one might surmise that a few cases wrongly diagnosed as de Quervain's disease have been cured by such forcible methods.

REFERENCES

SCHNEIDER, L. C. (1928), *Surg. Gynec. Obstet.*, **66**, No. 6, 846.

ZADEK, I. (1942), *J. Bone Jt Surg.*, **24**, No. 2, 37.

AN UNUSUAL CASE OF SUPPURATIVE HEPATITIS DUE TO STREPTOCOCCUS VIRIDANS

RECOVERY WITH PENICILLIN AND SULPHATHIAZOLE

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THE following case is thought worthy of record because it presents many points of interest to the clinician, the pathologist, and the bacteriologist.

CASE REPORT

HISTORY.—The patient, a tram conductress aged 26 at the time of the present admission, had already had a chequered career in hospital. Dyspepsia began at the age of 16, and a year later in 1936 she was treated for a gastric ulcer in a medical ward. Laparotomy was carried out in 1938, but the projected gastrectomy was abandoned because of the technical difficulty of dealing with a very high gastric ulcer which was penetrating the pancreas. She was re-admitted in 1939 with a severe hæmatemesis. A free perforation into the general peritoneal cavity was closed in January, 1945. She was re-admitted two months later because of severe and continuous pain. A high subtotal gastrectomy was performed on March 21, 1945. An operative detail, which appears important in retrospect, is that the ulcer crater (after cauterization with pure carbolic) was perforce left embedded in the pancreas. Convalescence was uneventful, and the patient returned to full work as a tram conductress after six weeks. She remained at full work for a further six weeks, free of all dyspepsia, and feeling splendidly fit till the onset of the present illness.

She was re-admitted (June 17) on the fifth day of illness. She said that on the first day she had felt nauseated when she awakened, but had gone to work. At 10 o'clock pain in the back had begun "suddenly", but not so suddenly as on the occasion when the ulcer had perforated. The pain was at first across the loins and not very severe, but as the day went on the severity increased and the pain spread upwards over the lower ribs behind. Despite increasing pain and increasing nausea she persisted at work till the evening, when she yielded and went home, and later in the evening she vomited bilious material for the first time. Pain in the back and frequent vomiting continued till admission. Feverishness and sweating began on the second day of illness, but at no time did she have a rigor. On the third day she noticed a "lump" in the left hypochondrium, and this gradually enlarged.

ON EXAMINATION.—On admission she was clearly desperately ill. She was flushed, sweating, writhing from side to side, and moaning with pain. The temperature was 103° F., and the pulse was 130 per minute, and of poor quality. On examination of the abdomen the most arresting feature was the presence of a visible tumour in the left hypochondrium the size of a grape-fruit. Its contours were smoothly rounded, and its central part was elevated about an inch above the general level of the abdomen. It occupied the whole of the left hypochondrium from the costal margin to the level of the umbilicus, and it transgressed the midline for an inch to the right. It moved freely on respiration. On palpation, the rounded margins were easily defined except above, where the tumour extended under the ribs. The tumour was of a uniform consistence, firm and elastic

and tenderness was strikingly absent. As for the rest of the abdomen, respiratory movements were unrestricted and there was no muscular guarding: the right lobe of the liver was not palpable. Examination of the loins, which the patient indicated as the site of pain, revealed neither tenderness nor guarding, and there was no tumidity.

The correct diagnosis was not made. It was clear that the patient was desperately ill from an inflammatory lesion, and it was conjectured that a localized abscess had formed as the result of perforation of an anastomotic ulcer which was walled off by adhesions. Laparotomy was carried out with the object of draining the abscess.

LAPAROTOMY (June 17).—The tumour was found to be the grossly enlarged left lobe of the liver. Visible beneath the serous covering was an uncountable multitude of abscesses each the size of a pin's head. The right lobe of the liver was also enlarged, but not so grossly as the left. It could not be freely inspected because of numerous adhesions: these were successively divided until the right lobe had been inspected for a distance of about an inch to the right of the gall-bladder. All surfaces of the left and right lobes so exposed were closely beset with miliary abscesses. The liver substance between the abscesses were engorged with blood and plum-coloured. Despite the risk of severe hæmorrhage, a deep section of the left lobe was removed for biopsy. Hæmorrhage was profuse, and could be only partially controlled by a gauze pack, round which the wound was closed.

Inspection of the slice of liver removed for biopsy showed at once that closely-set abscesses, each oozing a bead of pus, were scattered throughout the depth of the section. Examination of the pus revealed the presence of many streptococci, present in short chains up to 6 or 8 cocci.

At this time the chance of recovery appeared to be very slight. The only possibility of effective treatment seemed to lie in heavy dosage with penicillin reinforced with sulphonamide. Accordingly the administration of penicillin was begun at once without waiting to verify that the organism was penicillin-sensitive.

DOSAGE OF PENICILLIN AND SULPHONAMIDE.—

a. Penicillin.—

	By Intramuscular Drip continuously over Each 24-hour Period	*By Intravenous Drip, Each Dose given over a 4- to 5-hour Period*
1st day	400,000 units	500,000 and 500,000 units
2nd day	400,000 units	500,000 and 500,000 units
3rd day	200,000 units	500,000 units
4th day	200,000 units	500,000 units
5th–20 days	400,000 units	(None)

Total penicillin about 10,000,000 units.

b. Sulphonamide.—During the first 24 hours, 20 c.c. of soluseptasine were given intravenously every six hours. Thereafter, this was replaced by sulphathiazole by mouth, the dosage being 2 g. at 6 a.m. followed by 1 g. four-hourly till 10 p.m., none of the drug being given at night. This was continued till the sixteenth day, a total of some 90 g. being administered.

A check was kept on the bacteriostatic activity of the blood against the standard Oxford strain of *Staph. aureus*.

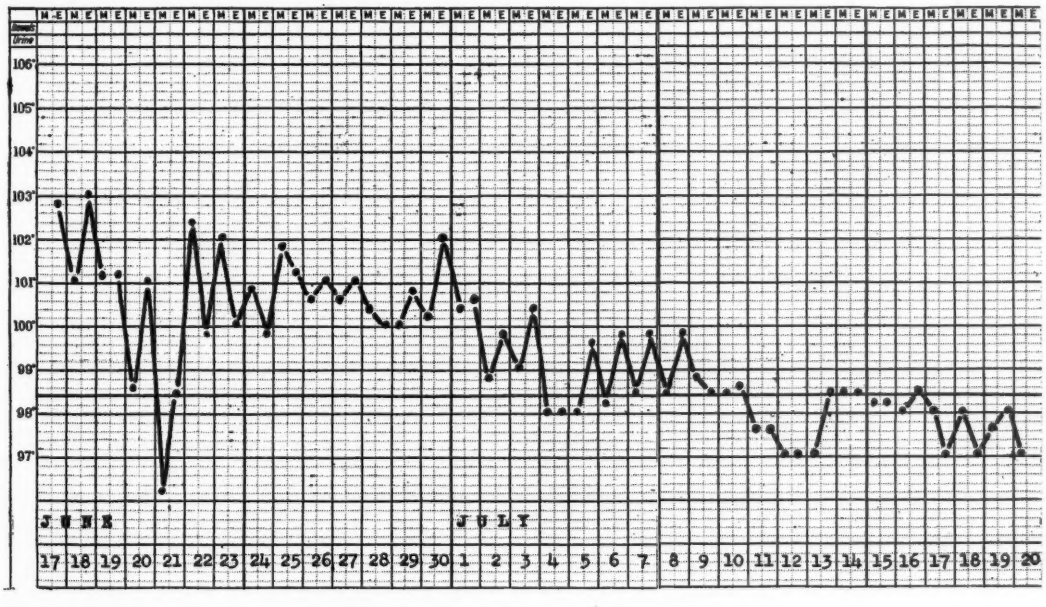

FIG. 286.—Axillary temperature-chart from admission on fifth day.

The bacteriostatic activity was disappointingly low, considering the high dosage of penicillin: the following values are representative:—

1. Evening of fourth day: total inhibition 1 : 1
   partial inhibition 1 : 2
   (The dosage was for this reason increased on the fifth day.)
2. Fifth day: total inhibition 1 : 2
   partial inhibition 1 : 4
3. Eighth day: total inhibition 1 : 1
   partial inhibition 1 : 4

Blood-culture on the fifth day was sterile.

PROGRESS.—Recovery was slow, but progress was steadily maintained. Thus the temperature fell by gradual lysis over a period of three weeks (*Fig.* 286). Slight improvement in the general condition was noted on the third day, and on the fifth day the patient began to take an interest in her surroundings. Her appetite returned at the end of a week and she began to take light diet. After three weeks she was eating well and sleeping without sedatives. She was allowed home on the

twenty-ninth day. A few of the details of her convalescence are of sufficient interest to be recorded.

Vomiting continued during the first four days, becoming progressively less troublesome. During this period intravenous saline and glucose had been given, but were stopped on the fourth day: that evening it was noted that there was a little œdema of the ankles. During the next few days her thirst was unquenchable, and she drank copiously. On the fifth and sixth days she developed gross œdema of both legs, and a gross acute ascites. During the next fortnight the œdema and ascites gradually diminished, and at the time of her discharge from hospital there was no more than the slightest pitting on pressure above the ankles.

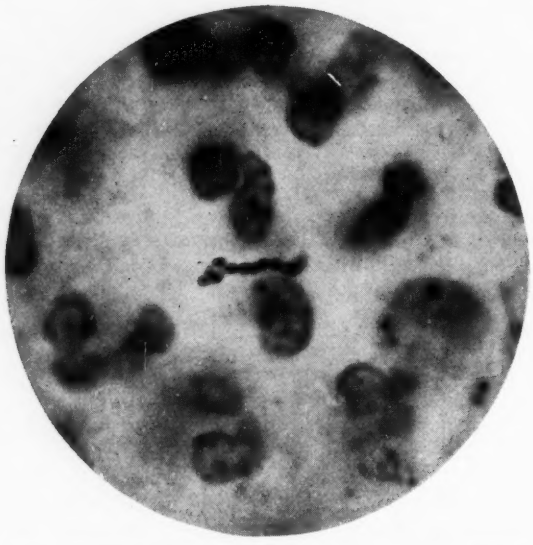

FIG. 287.—Section cut from fixed tissue, stained by Gram's method, to show the streptococcus. (× 2300.)

FIG. 288.—Masson's stain. Showing the great number of minute abscesses, each surrounded by a darkly stained ring of fibrinous exudate. (× 15.)

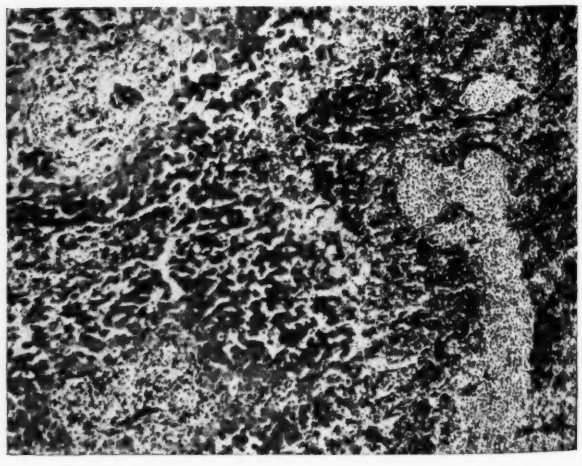

FIG. 289.—Masson's stain. Showing an abscess with surrounding fibrinous exudate on the right, and increased cellularity of two portal tracts on the left. (× 60.)

Pain across the loins remained troublesome during he first ten days. On the tenth day she complained for the first time of pain in the left hypochondrium. This lasted for only a few days, and by the seventeenth day all pain ceased.

At no time was the patient jaundiced, and it is noteworthy that throughout the illness rigors were absent, save following two abortive attempts to give a transfusion of blood.

SOURCE OF INFECTION.—The source of the infection remains unproved, but there appears to be good reason to incriminate the floor of the gastric ulcer (which, it will be remembered, was left embedded in the pancreas at gastrectomy three months previously). It is unlikely that the organism reached the liver by the bile-ducts or by the hepatic artery. Thus an ascending biliary infection is improbable in the absence of any history suggesting cholecystitis and in view of the fact that a normally functioning gall-bladder has since been demonstrated by a cholecystogram series. Again, if the infection had originated in a dental abscess or an endocardial vegetation and had reached the liver by the systemic circulation, some manifestation of infection of other organs might have been expected. But at no time were there painful subcutaneous nodes, nor were red blood-cells to be found in the urine on repeated examinations. Furthermore, it is difficult to conceive of a systemic septicæmia heavy enough to give rise to such countless numbers of minute abscesses which by their uniform size indicated a common age. Indeed, rupture of an abscess into a tributary of the portal vein seems to be the only plausible explanation and such an abscess might well have been present in the pancreas, originating in the embedded gastric ulcer.

NATURE OF THE ORGANISM.—In direct smears of the pus from the biopsy specimen, and in actual stained sections (*see Fig.* 287) the only organism seen was a streptococcus occurring in chains of 6–8 cocci. This organism on culture gave a pure growth of *Str. viridans*, exhibiting the classical properties of that organism. Thus it proved to be an aerobe, producing green pigment on human or horse blood, and dark-green pigment on chocolate agar. It produced acid in lactose, glucose, sucrose, and inulin, and acid and clot in milk. It did not ferment mannite and it did not survive heating at 56° C. for 30 minutes: these features eliminate any possibility of confusion with an atypical strain of the enterococcus group. The organism was shown to be penicillin-sensitive.

It is a matter for comment that this organism, whose aggressiveness is of a low order, should as an unmixed infection invade healthy vascular tissue and be strongly pyogenic. The organism is common in such mixed infections as dental or tubo-ovarian abscesses, but when it occurs alone as in endocarditis or in meningitis the formation of pus is either absent or scanty. Perhaps the explanation of its behaviour in the present case lies in the mode of origin of the infection. The postulated origin by rupture of an abscess into the portal stream might give rise to such an overwhelmingly heavy infection

that even an organism of low aggressiveness might be able to establish itself.

HISTOLOGY.—The outstanding feature of the stained sections is the multiplicity of small abscesses separated by narrow bands of compressed liver cells. *Fig.* 288 illustrates a field representative of the whole section. It is indeed remarkable that a liver whose structure is so extensively disorganized could support life, and be compatible with a return to abounding health. The section has been stained by Masson's method to show the ring of fibrin surrounding the abscesses. *Fig.* 289 illustrates the appearance at a magnification of × 60, and in addition to one frank abscess shows aggregation of polymorph leucocytes round the portal systems.

LIVER FUNCTION.—It was apparent that observations on hepatic function would be of interest, and the tests carried out are shown in the following table.

	JUNE 22	JULY 11	AUG. 17	OCT. 9
Plasma bilirubin (mg. per cent)	0·5	0·2	0·2	0·2
Glucose tolerance	Normal	—	—	—
Lævulose tolerance	Abnormal	High normal	Abnormal	Normal
Cephalin flocculation	+++	+++	++	Negative
Albumin (g. per 100 ml. of plasma)	2·68	3·76	4·14	4·63
Globulin (g. per 100 ml. of plasma)	2·43	3·62	2·49	2·21
Fibrinogen (g. per 100 ml. of plasma)	0·50	0·46	0·32	0·32
Total protein (g. per 100 ml. of plasma)	5·61	7·84	6·95	7·16
Packed cell volume (per cent)	22·0	31·8	34·0	—

It is noteworthy that the plasma-bilirubin level was at no time above normal. Glucose tolerance estimated on June 22 showed no abnormality. The lævulose tolerance tests, interpreted according to Rennie (1943), suggest impairment of liver function on the first three occasions of testing. The cephalin flocculation test, introduced by Hangar (1939) has been chiefly used to detect damage to the liver parenchyma: flocculation (i.e., a positive result) was present on the first three occasions, but absent on the last.

The alterations in the plasma-protein levels are somewhat difficult to interpret. Disease of the hepatic parenchyma and infective processes are alike associated with a fall in plasma-albumin and a rise in plasma-globulin. In this patient when suppuration was present actually in the liver, pronounced alterations would be expected. Lowered albumin and raised globulin values were indeed present on July 11, but not on the first occasion, when the albumin was low but the globulin level appeared to be normal. Probably both estimates on this occasion were fictitiously low because of the hæmodilution (evidenced by the low packed cell volume) which accompanied the œdema already noted at this time. The third and fourth estimations show that the albumin and globulin levels gradually returned to normal.

LATER PROGRESS AND PROGNOSIS.—After discharge from hospital the patient continued to make good progress. She returned for examination on Aug. 17 (three months after the onset of suppurative hepatitis). She reported that she was in excellent health and was free from indigestion. She looked a normal young woman, and had acquired a healthy layer of subcutaneous fat. No abnormality was detected on examination, and the liver was not enlarged. She returned to work as a tram conductress at the end of August, and in January, 1946, was still continuously at work and in excellent health.

It was feared, at the time the patient left hospital, that contracture of scar tissue in the liver might lead to obstruction of the portal circulation and ascites. Full health and normal hepatic function tests have, however, been maintained for seven months, and it now appears safe to conclude that recovery is to be complete.

It is a pleasure to express my gratitude to Professor Illingworth for his stimulating interest in this case and to Doctors Rennie, Lominski, and Trounson of the Departments of Medicine, Bacteriology, and Pathology for their technical reports and valuable suggestions. I am further indebted to Dr. Trounson for the illustrative microphotographs.

REFERENCES

HANGAR, F. M. (1939), *J. clin. Invest.*, **18**, 261.
RENNIE, J. B. (1943), *Brit. J. exp. Med.*, **24**, 26.

CASE OF LYMPHANGIOMA IN THE REGION OF THE HEAD OF THE PANCREAS CAUSING AN OBSTRUCTIVE JAUNDICE

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A CASE of obstructive jaundice simulating carcinoma of the head of the pancreas clinically but caused by an unusual simple tumour is described.

CASE REPORT

The patient, Mr. R., aged 66, was admitted to Chalmers Hospital on Sept. 6, 1941. On admission, the patient was deeply jaundiced, rather cachectic, and had obviously lost a great deal of weight. He thought that he had been losing weight for about five months, and he had also suffered from a little flatulence, loss of appetite, and heartburn, but had had no sickness or pain. A fortnight before admission his doctor noticed that he was jaundiced. This became progressively more severe.

ON EXAMINATION.—There was a large palpable gall-bladder, and below this a large, firm, palpable tumour. The patient was such a typical case of carcinoma of the head of the pancreas so far advanced that we very nearly considered leaving him alone. He was, however, very miserable and had a great deal of itch and discomfort, and in view of this it was decided to carry out some palliative procedure.

AT OPERATION.—The gall-bladder was found to be very distended and there was a large tumour about the size of a cricket ball in the region of the head of the pancreas. This did not have quite the stony-hard feeling of a carcinoma, nor could any secondaries be felt. A small piece was taken for biopsy and a cholecystgastrostomy was performed.

The patient's jaundice immediately began to improve and his general condition became better. The section

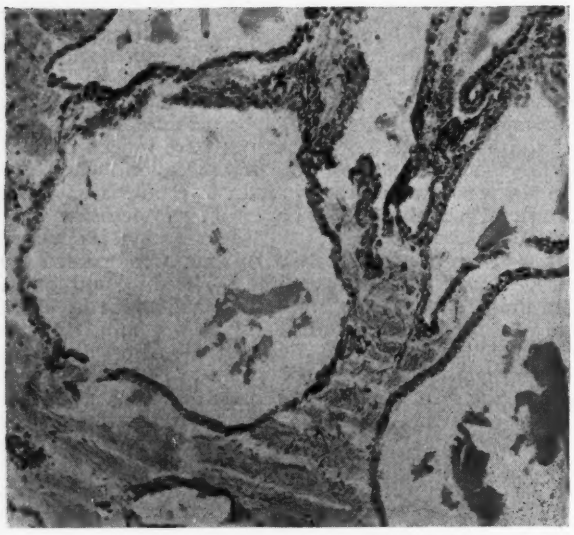

FIG. 290.—Section of tumour.

of the tumour (*Fig.* 290) showed a lymphangioma with multilocular cavernous spaces and endothelial lining, and there was some perivascular endothelial increase with no inflammatory reaction. There was no evidence of malignancy. Since excision of the tumour would have been exceedingly hazardous, it was decided to treat it with deep X-ray therapy.

Deep X-ray therapy was administered by Dr. McWhirter, Royal Infirmary, Edinburgh, from Dec. 4 to Feb. 16, 1942, the dose being 3000 r.

The patient progressively improved from the time of his operation, and as it was rather difficult to assess the value of the X-ray therapy, a barium meal was given before the treatment. *Fig.* 291 shows the state of affairs on Nov. 6, 1941. It will be noticed that there is very marked broadening of the D of the duodenum, the first part being pushed upwards and the third part downwards by the tumour. A subsequent barium meal (*Fig.* 292) carried out on Nov. 27, 1945, shows the duodenum to have returned to its normal position and barium can be seen entering the gall-bladder.

Since his operation and treatment, the patient has continued to be in good health.

FIG. 291.—Radiograph following a barium meal, showing condition on Nov. 6, 1941. Marked broadening of D of duodenum and change of position, the first part being pushed upwards, the third part downwards, by the tumour.

FIG. 292.—The appearance on Nov. 27, 1945, showing the return of the duodenum to its normal position.

A CASE OF A CHRONIC VOLVULUS OF THE JEJUNUM DUE TO MULTIPLE JEJUNAL DIVERTICULA

By H. W. PORTER

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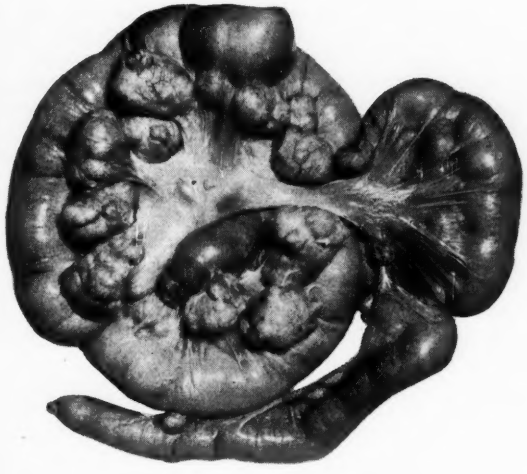
WHILE diverticula of the jejunum and ileum are not exceedingly rare conditions, especially of the jejunum, it is unusual for them to give rise to symptoms, and it is thought that it might be of interest to record a case with a chronic volvulus. The complications associated with diverticula of the jejunum may be: (1) Inflammatory disturbances giving rise to perforation; (2) Occasional intestinal hæmorrhage; (3) Rupture, which may be spontaneous or traumatic; (4) Foreign bodies causing perforation; (5) Neoplasms or neoplastic disease which occasionally takes place in the diverticula; (6) Acute intestinal

obstruction or sometimes, but rarely, chronic intestinal obstruction. Complications arising from diverticulosis of the jejunum are uncommon. Dickson and Waugh (1943) described symptoms in only 13 of 122 cases recorded at the Mayo Clinic.



FIGS. 293, 294.—The portion of jejunum resected, showing the twisted loops and the numerous diverticula.

Walker (1945) states that volvulus causing acute intestinal obstruction has only been recorded twice, but records no case of chronic volvulus.

A case of chronic intestinal obstruction due to a chronic volvulus associated with diverticulosis of the jejunum is described.

CASE REPORT

HISTORY.—The patient, a male aged 64, was admitted to Chalmers Hospital in August, 1944, complaining of pain in the umbilical and epigastric regions, from which he had been suffering periodically for two years. The pain at that time was colicky in nature. Though sometimes very severe, it was relieved with antispasmodics, and latterly it had become increasingly worse and the patient began to complain of waterbrash and heart-burn. Vomiting became more frequent, until he was vomiting every 24 hours. The vomiting was sometimes induced by himself in order to relieve flatulence, and the vomitus occasionally contained a little blood. The patient's pain became worse immediately after meals and was sometimes relieved by lying down. He was admitted to hospital and a barium series was rather inconclusive and showed rather a large stomach and a slight deformity of the duodenal cap. There was no evidence in the barium series of diverticulosis. The patient had a high rising gastric acidity and he was treated medically with diet and rest in bed. With this he improved considerably and was discharged. He remained well except for occasional bouts of colic and distension until July, 1945, and put on weight.

During the intervening period he was considerably constipated and for this he took a regular aperient. In July, 1945, three weeks before his second admission to hospital, he had a sudden attack of abdominal pain, nausea, and vomiting, and on this occasion the vomitus was quite definitely dark in colour. Immediately after this, he had a recurrence of his old symptoms, intermittent colicky pain, repeated vomiting, loss of weight, flatulence, and a degree of constipation. He was re-admitted to hospital and a further barium meal again revealed no definite peptic ulcer, nor did it show any abnormality in the small intestine. His test-meal again showed rather a high acid value, he had occult blood in his stool, and there was considerable loss of weight. Since he was not responding to medical treatment, it was decided to perform a laparotomy.

AT OPERATION.—The stomach was found to be rather distended and there was some scarring at the pylorus, with a small posterior duodenal ulcer, and it was decided to do a posterior gastro-jejunostomy. On seeking the jejunum, it was found that the first two loops were distended, hypertrophied, and had an exceedingly long mesentery. Both loops were twisted through 180° on the mesentery and were the site of numerous diverticula. The diverticula started about 4 in. below the duodenojejunal junction, were largest at the upper end, and then gradually tailed off, and about 3 ft. farther down the jejunum became normal. No diverticula were found in the ileum, colon, or duodenum. The affected portion of jejunum was resected and an end-to-end anastomosis performed, following which a posterior iso-peristaltic gastrojejunostomy was carried out. (*Figs.* 293, 294.)

The patient had an uneventful convalescence and has had complete relief from his symptoms.

REFERENCES

BENSON, R. E., DIXON, C. F., and WAUGH, J. M. (1943), *Ann. Surg.*, **118**, 377.

WALKER, R. MILNES (1945), *Brit. J. Surg.*, **32**, 457.

AN UNUSUAL CASE OF SHARK-BITE

By ERIC DAVIES
SURGEON, ANGLO-IRANIAN OIL CO., LTD.

NINE cases of shark-bite have been operated upon during the last two years, of which 7 were partial or complete amputations of arm or leg.

The ninth case is reported in detail as it is unusual and illustrates the remarkable protective functions of the omentum.

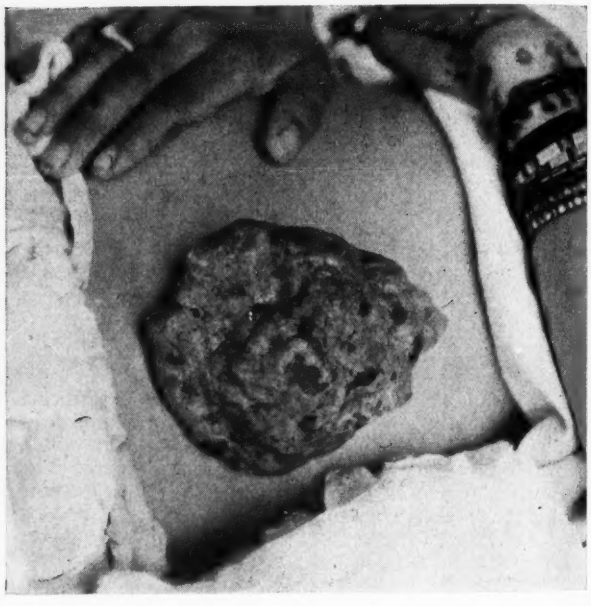
CASE REPORT

HISTORY.—The patient, an Arab woman of 30, was a passenger on a dhow which was wrecked at the head of the Persian Gulf. She had been holding on to a piece of floating wreckage for an hour when she was attacked by a shark, which wounded her in the abdomen but did not succeed in pulling her down. She was in the water for a further four hours and was then rescued and put ashore at a small Arab village. The rescue work was directed by an English pilot who was under my care some months later and was able to verify the times and dates, and from whom it was learnt that over 40 persons were lost in the wreck, mostly due to sharks.

The patient remained in the village for five days, during which time the mass on her abdomen was kept covered by a damp cloth. As she showed no signs of dying the inhabitants brought her to Abadan in a lorry, the journey taking 24 hours owing to a breakdown in the desert.

ON EXAMINATION.—She was a fit-looking woman much decorated with tattoo marks and bracelets. Her temperature was 98·4° and pulse 72, her tongue was clean, and she had not vomited. Her bowels had acted once since the accident. On the left side of the abdomen was a large mass protruding through a transverse wound in the abdominal wall, it being quite soft and pearly grey in colour, and gurgling sounds could be obtained by pressure (*Figs.* 295, 296). There was slight guarding in the vicinity of the wound, but the remainder of the abdomen was quite soft and there was no distension or shifting dullness. There was no tenderness on rectal examination.

OPERATION.—The surface of the mass was cleaned with saline and a line of cleavage found which made easy the division of the wall. This consisted of omentum quite ½ in. thick and contained a loop of transverse colon covered by smooth shining peritoneum. The outer surface of the omental sac was separated from the tissues of the abdominal wall by gauze dissection and slight traction applied so that the extra-abdominal portion could be resected after securing the vessels by multiple ligatures. The bowel could then be replaced through the wound, which was found to be 4½ in. long at the skin level and 2½ in. at the peritoneum. Part of the adjoining descending colon could be seen and showed no signs of bruising and there was no adherent small intestine. A corrugated rubber drain was inserted through the wound, which was closed by all-layer sutures.



FIGS. 295, 296.—Photographs of patient, showing site and extent of abdominal protrusion.

PROGRESS.—The drain was removed after 24 hours and the wound healed well, her recovery being uneventful. She was free of symptoms when seen one month later and there was no weakness at the site of the bite.

Comment.—It seems remarkable that a wound of this size could be caused by a shark's tooth without causing visceral damage. It is also interesting to speculate whether the four hours' immersion in sea water was a factor in producing the complete covering of omentum.

REVIEWS AND NOTICES OF BOOKS

Regional Analgesia. By H. W. L. MOLESWORTH, F.R.C.S. (Eng.), Senior Surgeon, Royal Victoria Hospital, Folkestone. Second edition. $8\frac{5}{8} \times 5$ in. Pp. 92 + viii, with 42 illustrations. 1946. London: H. K. Lewis & Co. Ltd. 8s. 6d. net.

FIRST published in 1940, the second edition of this small book contains few alterations or additions, and remains an excellent account of the personal experiences of the author in the use of regional analgesia.

The text is well arranged and interesting to read, and the numerous illustrations are well produced and achieve their object.

Modern Anæsthetic Practice. *The Practitioner Handbooks.* Edited by the late Sir HUMPHREY ROLLESTON, Bt., G.C.V.O., K.C.B., M.D., F.R.C.P., and ALAN MONCRIEFF, M.D., F.R.C.P. Second edition. $8\frac{1}{2} \times 5\frac{1}{4}$ in. Pp. 150 + viii, with 7 illustrations. 1946. Eyre & Spottiswoode (Publishers) Ltd., for *The Practitioner*. 12s. 6d. net.

IN the editorial preface to the second edition we read: "Those in general practice are constantly being required to give anæsthetics, and it is for them rather than for the specialist-expert that this book was planned and for whom, once again, the second edition is intended. If it continues to be found of value then it is to the skilled contributors that thanks are due, and editorial gratitude must be added for their cheerful co-operation, suggestions, and advice."

In our opinion the objects of this book have been largely achieved, for it contains a wealth of information and the contributions are well arranged and form a genuine synopsis of modern anæsthetic practice.

At a time when the ever-increasing mechanization of practical anæsthesia is receiving a good deal of criticism, it is interesting to read the comments of Dr. John Gillies at the commencement of Chapter II: "The volatile anæsthetic drugs still form the main stock in trade of the occasional or less experienced anæsthetist. No elaborate equipment is necessary for their application and therefore in this respect they are simple as well as convenient and portable. The anæsthetist should appreciate, however, that the advantages just mentioned accrue to himself rather than to the patient, and that in simple technique there are undesirable features which he must always keep in mind and which it is the purpose of modern apparatus to eliminate, or at least to modify."

In Chapter IV, on Basal Narcotics, we note that in the discussion on complications of pentothal no mention is made of the use of picrotoxin, or the dangers of intra-arterial injection. This last complication is a catastrophe, and every occasional anæsthetist should be instructed how to avoid it.

In Chapter VII, on Anæsthesia and Analgesia in Midwifery, the contributor goes out of his way to emphasize the value of chloroform in domiciliary obstetrics, and endeavours to make a case for the continued use of this drug. In fact, he deplores the lack of routine instruction in teaching hospitals, and even attempts an analogy to road mortality: He writes: Speed in itself may be dangerous, but it is not speed alone which kills, but impatience and lack of due care and attention. Speed in the wrong place kills, so do the various anæsthetic agents. The need is to learn where those wrong places are."

In our opinion this contributor is defending and advocating a lost cause, and we consider the views of Dr. Paluel Flagg, published in a recent text-book, to be a much better expression of contemporary obstetric and anæsthetic opinion. Flagg writes: "Chloroform, while ideal in efficiency, is a dangerous poison. In the light of present-day pathology, chloroform should cease to be used as an anæsthetic in obstetrics."

This book is well produced, the illustrations are clear and adequate, and the volume should continue to be a popular contribution to the "Practitioner Handbook" library.

The Care of the Neurosurgical Patient. Before, During, and After Operation. By ERNEST SACHS, A. B., M.D., Professor of Clinical Neurological Surgery, Washington University School of Medicine, Saint Louis. $9\frac{1}{2} \times 6\frac{1}{2}$ in. Pp. 268, with 177 illustrations. 1945. London: Henry Kimpton. 30s. net.

THIS is an important book which deals with an important subject. Professor Sack's extensive and ripe experience is set out in a most interesting and fascinating manner. In this monograph the important points dealing with the care of the neurosurgical patient before, during, and after operation are given in a very practical way.

The numerous illustrations are of great help to the reader and enhance the value of the book.

The various neurosurgical methods used by the author are the outcome of a life-long study of this branch of surgery and are ones which have stood the test of time.

Neurosurgeons the world over will read this book with interest and profit.

The Anatomy of the Bronchial Tree. With Special Reference to the Surgery of Lung Abscess. By R. C. BROCK, M.S. (Lond.), F.R.C.S. (Eng.), Surgeon to Guy's Hospital; Surgeon to the Brompton Hospital; Surgeon to an E.M.S. Thoracic Surgical Centre. $9\frac{3}{8} \times 6$ in. Pp. 96 + vi, with 142 illustrations, many as plates. 1946. London: Geoffrey Cumberlege, Oxford University Press. 42s. net.

THIS monograph is in reality a reprint of an excellent series of articles published in the *Guy's Hospital Reports* during recent years. The advent of bronchoscopy and modern X-ray pictures has added enormously to our knowledge of the anatomy of the bronchial tree, and Mr. Brock has increased our understanding in the surgery of lung abscess.

This book will prove of real value, not only to surgeons and physicians who are interested in the diseases of the chest, but also to anatomists and physiologists.

The illustrations throughout the book are of a high order of perfection.

Anatomical Eponyms. Being a Biographical Dictionary of those Anatomists whose Names have become incorporated into Anatomical Nomenclature, with Definitions of the Structures to which their Names have been attached and References to the Works in which they are described. By JESSIE DOBSON, B.A., M.Sc. With a Foreword by Professor F. WOOD JONES. $8\frac{1}{2} \times 6\frac{5}{8}$ in. Pp. 240 + x, with Frontispiece. 1946. London: Baillière, Tindall & Cox. 30s. net.

RECENT standardization of anatomical nomenclature has resulted in suppressing or limiting the traditional and, on the whole, desirable custom of linking the names of the original workers with the anatomical structures which they discovered. Apparently in Great Britain—says the author—we are now committed to a system of anatomical terminology from which all eponymous nomenclature has been removed.

This does seem a pity, and so unnecessary. The author apparently feels it so strongly as to justify this biographical dictionary of those anatomists whose names have become associated with certain structures. The book, which is beautifully got up, has a Foreword by Professor Wood Jones and a Frontispiece of Vesalius. It is a very useful book of reference.

Patients and Appendicitis. By Sir CRISP ENGLISH, K.C.M.G., F.R.C.S., Consulting Surgeon, St. George's Hospital; Hon. Consulting Surgeon, Queen Alexandra's Military Hospital, Millbank; etc. 8⅜ × 5⅜ in. Pp. 155 + vii. 1946. London: J. & A. Churchill Ltd. 10s. 6d.

THIS is an interesting little monograph which is full of sound surgical common sense and it deals with a somewhat neglected subject.

How often the patient is neglected and not taken into account by the doctor or surgeon concerned when the question of operation for appendicitis is considered.

Sir Crisp has drawn from his matured and extensive clinical experience and acumen in the writing of this book. It should prove of value, not only to the practitioner, but also to the student, who will learn more from this little book about appendicitis than he will from the average text-book on surgery.

A concise, up-to-date, and useful bibliography on appendicitis is given at the end of the volume.

Manual of Diagnosis and Management of Peripheral Nerve Injuries. By ROBERT A. GROFF, M.D., and SARA JANE HOUTZ, B.S. With an Introduction by I. S. RAVDIN, M.D. 8⅞ × 6 in. Pp. 188 + xii, with 111 illustrations. 1946. Philadelphia and London: J. B. Lippincott Co. 36s. net.

THIS is a practical little monograph which gives a new approach to the diagnosis, management, and after-care of peripheral nerve injuries.

The book is divided into two parts. Part 1 is devoted to a general discussion of nerve injuries, with specific advice on how to recognize immediately, and manage effectively, injuries affecting the peripheral nerves. Part 2 presents an illustrated outline of peripheral and cranial nerve function. Muscle function is shown effectively by action diagrams.

There are over a hundred illustrations, quite a number of which are original and most instructive.

There are a few mistakes which require correction—on page 136, the deep peroneal nerve is stated to supply the interossei, which is quite wrong.

The book should prove of value to general practitioners, surgeons and neurosurgeons, and physiotherapists who require a quick reference book which can be used in the out-patient department or ward.

Penicillin. Its Practical Application. Under the General Editorship of Professor Sir ALEXANDER FLEMING, M.B., B.S., F.R.C.P., F.R.C.S., F.R.S., Professor of Bacteriology in the University of London, St. Mary's Hospital, London. 8½ × 5½ in. Pp. 380 + xi, with 59 illustrations. 1946. London: Butterworth & Co. (Publishers) Ltd. 30s. net.

ALTHOUGH much has been written concerning penicillin during the last three years, yet there is room for this volume. Sir Alexander Fleming, who is the general editor and has written about the history and development of penicillin, and the bacteriological control of penicillin therapy, has collected a band of expert clinicians who have written concise practical articles dealing with their specialities. Porritt and Mitchell have contributed a well-balanced article on the prophylactic use of penicillin and quite rightly emphasize the important fact that penicillin does not replace surgery. Vaughan Hudson writes on generalized infections and reports some interesting clinical cases which were treated with penicillin. Hope Gosse deals with chest infections, while Holmes Sellors contributes a chapter on Chest Surgery. Dickson Wright has written an excellent chapter on the value of penicillin in infections of the brain, while McElligott deals with the problems of venereal diseases.

The book is an important one, as it gives the latest practical details of penicillin therapy. No surgeon can afford to be without this really useful book.

Pathology in Surgery. By N. CHANDLER FOOT, M.D., Professor of Surgical Pathology, Cornell University Medical College; Surgical Pathologist, New York Hospital. 10 × 6⅞ in. Pp. 511 + xi, with 368 illustrations in text and 11 coloured plates. 1946. Philadelphia and London: J. B. Lippincott Co. 60s.

THIS is a book on surgical pathology written by a pathologist, and, when all is said and done, this is the right type of author. Some members of the profession consider that a book on surgical pathology should be written by a surgeon, but Professor Foot has easily refuted this idea by the publication of this book. He establishes the significant place occupied by pathology in surgery, and draws a precise boundary between it and pathology in general.

The book is profusely illustrated with some 400 microphotographs and some coloured photographs. The microphotographs are excellent, they are clear and well reproduced, but it is a pity that the magnification is not given in each case. Surgical pathology to-day covers a vast field and this book includes the cardiovascular system, blood-forming organs, respiratory system, alimentary tract, liver, gall-bladder, pancreas, urinary system, organs of internal secretion, the breast, nervous system, and male and female reproductive system.

There can be no doubt that this book will prove of real value to surgeons who require a reference volume on surgical pathology.

The Principles and Practice of War Surgery. With Special Reference to the Biological Method of Treatment of Wounds and Fractures. By J. TRUETA, M.D., Hon. D.Sc. (Oxon.), Surgeon (E.M.S.), Wingfield-Morris Orthopædic Hospital, Oxford; Acting Surgeon-in-Charge, Accident Service, Radcliffe Infirmary, Oxford; Formerly Director of Surgery, General Hospital of Catalonia, University of Barcelona. Third edition, revised and enlarged. 9⅛ × 6⅛ in. Pp. 426 + xvii, with 156 illustrations. 1946. London: William Heinemann Medical Books Ltd. 42s. net.

THAT a third edition of this book is required only three years after the publication of the first edition is evidence enough of its popularity and usefulness. Although we are back to times of peace, yet it is an era of mechanical transport, with its heavy toll of accidents causing lacerated injuries to children and adults alike, and the principles set out in this volume will be of value to the practitioner and surgeon who will have to deal with the civilian casualties.

Like its predecessors, this third edition is concerned in large part with those methods (i.e., wound excision and the complete encasement of wounded limbs in plaster) which are now generally associated with the author's name.

The book is well illustrated with excellent photographs and drawings, many of which help to elucidate the text. It will retain its place as an authoritative reference work.

The Management of Fractures, Dislocations, and Sprains. By JOHN ALBERT KEY, B.S., M.D., St. Louis, Wis., and H. EARLE CONWELL, M.D., F.A.C.S., Birmingham, Ala. Fourth edition. 9¾ × 6⅝ in. Pp. 1322, with 1316 illustrations. 1946. London: Henry Kimpton. 63s. net.

THE fourth edition of this well-known book on fractures has been carefully revised and brought up to date. War surgery has had something to give to orthopædic practice and this book contains the new methods in vogue at the present time for the treatment of compound fractures.

Skeletal traction is given its proper place in the treatment of fractures, and the danger of distraction is rightly emphasized. Perhaps the greatest advance that has been made is in the treatment of fractures of the jaw and face, as much has been learnt from the numerous cases which occurred during the recent world war.

This book is profusely illustrated with some 1310 clinical, radiological, and line drawings, but it is a pity that some of the older illustrations have not been replaced, as they are rather indistinct.

This volume still maintains its place as a reference volume dealing with traumatic surgery of the skeleton.

Penicillin in General Practice. By J. L. HAMILTON-PATERSON, M.D., Pathologist to Redhill County Hospital, Edgware. 6⅝ × 4 in. Pp. 95, with 10 illustrations. 1946. London: Staples Press Ltd. 5s. net.

THIS compact little volume would seem to have everything to recommend it. It is of handy size (to fit the pocket), well produced, cheap (at five shillings), and contains in its ninety-odd pages a wealth of clearly set out, practical information on a subject a more than cursory knowledge of which is quite reasonably to-day demanded by the patient of his doctor. The author, a pathologist, might be expected to stress the more scientific side of the subject, but while this receives very adequate consideration, it is obvious from a perusal of the book that Dr. Hamilton-Paterson mus have had very considerab e personal clinical experience of his subject-matter. In the first half of the book he deals with the properties, nature, and principles governing administration of penicillin; in the second half, with lines of treatment for various individual disorders. This latter half is remarkably comprehensive and includes brief chapters on the use of penicillin in venereal disease and the common dermatological, E.N.T., and eye diseases. Two good points are made—and remade—throughout the book. The first is that without suitable bacteriological control penicillin administration—now in the hands of such a vastly enlarged field—will easily fall into disrepute and lose that sound scientific basis on which its use was developed; and secondly that penicillin therapy cannot replace sound surgery where such is indicated, i.e., that dead tissue of all kinds is still inaccessible to penicillin and requires removal.

It is stated (pp. 16, 24) that synovial membranes of the larger joints form an impermeable barrier to the passage of systemic penicillin. Recent researches (Penicillin Therapy and Control, 21 Army Group) prove this is not the case, as sufficiently large parenteral dosage does produce easily recognizable penicillin concentration in the synovial fluid. The point is of minor practical importance, however, as local instillation into the joint cavity, as recommended by the author, is undoubtedly both more efficient and more economical. The chapter on the principles of treatment is eminently practical and the brief discussion on the time–dosage relationship is particularly to the point. It is to be hoped that the term a "course of penicillin" will not be allowed to creep into common usage. The only direction in which the book might be said to exceed its stipulated scope, and this perhaps a forgivable licence to a pathologist, is in the rather full discussion (and diagrams) of the somewhat cumbersome Eudrip apparatus and the complicated electric motor for systemic administration. These require adjustment and attention beyond the power or time of the average general practitioner, and mention might well be made of the use of the much simpler blood-transfusion bottle—to say nothing of a word on the value of the intelligent patient's co-operation during his waking hours in watching the drip-chamber.

But these are small criticisms of a most informative and accurate little book—one which it is felt will deservedly be most popular with those for whom it was primarily written, the general practitioners. Its field might with value be far more widely extended!

The Surgical Clinics of North America. Chicago Number, February, 1946. Symposium on Surgical Technic. 9 × 6 in. Pp. 269 + vi, with 172 illustrations. London and Philadelphia: W. B. Saunders Co. Annual subscription (six numbers), paper 55s.; cloth 75s.

THIS symposium on colon surgery is really the contents of the June, 1946, number of the Surgical Clinics of North America. Every aspect of colon surgery is discussed in the several interesting papers which go to make up this volume. The treatment of ulcerative colitis is given in a very comprehensive fashion, but the results in America do not seem any better than in this country.

The references are solely American.

Journal of the History of Medicine and Allied Sciences. Edited by GEORGE ROSEN. Volume I, Number 1, January, 1946. 10 × 6⅝ in. Pp. 183, illustrated. New York: Henry Schuman. Quarterly. $8.50; single copies, $2.50.

THE appearance of a new journal on the history of medicine, in the English language, must be regarded as a major event and commented on with care.

It is a matter for regret that the *Annals of the History of Medicine*, edited by Dr. Packard, became a war casualty and will not be revived. This new journal takes its place.

It may be wondered whether there is room for another similar publication alongside the *Bulletin of the History of Medicine*, edited by Professor Henry Sigerist. On this point it is stated that the policy of the new journal is that it will not compete with the *Bulletin*, but will supplement it.

The Editor of the new journal is Dr. George Rosen. He has written a first-class treatise on "The History of Miners' Diseases", and is reputed to be a first-class scholar. He says, in his introduction: "We do not want to cultivate medical history as a search for antiquities, as a hunt for curios, but rather as a vital, integral part of Medicine. We believe that this can be done." Yes. So do we! But we would like to say, very humbly, that the medical profession needs some coaxing to take any real interest in the history of our profession and that the process of instruction should not be too devastatingly dull.

This first volume contains 183 pages, with a number of illustrations, and among the ten or more subjects are: A substantial contribution by Professor Charles Singer on "Some Galenic and Animal Sources of Vesalius"; "The London Years of Benjamin Waterhouse", by J. C. Trent; and "A Note on William Blake and John Hunter", by Jane Oppenheimer.

The publisher is Henry Schuman, of New York, a publisher of sound repute, who has a remarkable knowledge of medical history. The printing and set-up of this initial volume is quite first class.

Cirugia estetica. By Dr. RAMON PALACIO POSSE. $9\frac{7}{8} \times 6\frac{7}{8}$ in. Pp. 299, with 187 illustrations. 1945. Buenos Aires: El Ateneo. Price not stated.

THE title of this South American Spanish book seemed strange and sent the reviewer to the *Oxford English Dictionary* to see how the word "æsthetic" could be applied to surgery. We are familiar with the term "Prosthetic Surgery", which is defined as "that part of surgery which consists of supplying deficiencies, as by artificial limbs, teeth, etc., 1706." But "Æsthetic Surgery"? The Dictionary says that "æsthetic" means: "of or pertaining to the appreciation of the beautiful", or, as an alternative meaning, "having or showing refined taste".

A survey of the book leads one to conclude that the term "Cirugia estetica" is meant to refer mainly to the surgical procedures required for the removal of blemishes and facial—mostly facial—deformities and irregularities, such as crooked noses, flapping ears, and wrinkles. Then there is also a long chapter on mammary hypertrophy and ptosis, with copious illustrations both before and after operation.

The whole work smacks of the beauty parlour rather than of academic surgery. Not that this type of work does not require the greatest skill. It does. And if anyone desires to do this type of work, this book should be of service.

Tumores broncogenicos. La Cirurgia toracica en los Estado Unidos de Norte America. By HERNAN D. AGUILAR, Buenos Aires. $8\frac{3}{4} \times 6\frac{3}{4}$in. Pp. 450 + x, with 12 illustrations and 7 plates. 1946. Buenos Aires: El Ateneo. Price not stated.

THIS book is based on the author's experiences in the study of bronchial tumours while spending two years in the United States, most of the time at the Barnes Hospital Chest Service, St. Louis, under Professor Evarts A. Graham. It is, therefore, basically an account of the work at Barnes Hospital and includes a study of experiences in 264 cases of bronchial tumours and 52 consecutive pneumonectomies. It is well worth consulting by those especially interested in lung diseases and in the results of treatment in this world-famous centre of thoracic surgery.

BOOK NOTICES

[*The Editorial Committee acknowledge with thanks the receipt of the following volumes. A selection will be made from these for review, precedence being given to new books and to those having the greatest interest to our readers.*]

Rœntgen Diagnosis of Diseases of the Gastro-intestinal Tract. By JOHN T. FARRELL, jun., M.D., Clinical Professor of Radiology, Graduate School of Medicine, University of Pennsylvania. 9 × 6 in. Pp. 271 + x, with 190 illustrations. 1946. Springfield, Ill.: C. C. Thomas. (London: Baillière, Tindal & Cox.) 30s.

Anesthesia in General Practice. By STUART C. CULLEN, M.D., Head of Division of Anesthesiology, Department of Surgery, State University of Iowa Hospitals; Associate Professor of Surgery (Anesthesiology), State University of Iowa College of Medicine. 8 × $5\frac{1}{2}$ in. Pp. 260, with 36 illustrations. 1946. Chicago: The Year Book Publishers Inc. (London: H. K. Lewis & Co.). 21s.

The Principles of Anatomy. An Introduction to Human Biology. By A. A. ABBIE, M.D., B.S., D.Sc. (Syd.), Ph.D. (Lond.), Elder Professor of Anatomy and Histology in the University of Adelaide. Second edition, revised and enlarged. $8\frac{1}{2} \times 5\frac{3}{8}$ in. Pp. 273 + x, with 76 illustrations. 1946. Sydney and London: Angus & Robertson Ltd. 12s. 6d. net.

The Surgical Clinics of North America. Lahey Clinic Number. Symposium on Colon Surgery. 9 × 6 in. Pp. 274 + vii, with 82 illustrations. London and Philadelphia: W. B. Saunders Co. Annual subscription (six numbers): papers 55s.; cloth 75s.

Surgery of the Hand. By R. M. HANDFIELD-JONES, *M.C.*, M.S., F.R.C.S., Surgeon to St. Mary's Hospital; etc. Second edition. $9\frac{5}{8} \times 6\frac{7}{8}$ in. Pp. 164 + xii, with 104 illustrations. 1946. Edinburgh: E. & S. Livingstone Ltd. 20s. net.

Diagnosis and Management of the Thoracic Patient. The American Practitioner Series. Edited by CHARLES PHILAMORE BAILEY, M.D. 9 × 6 in. Pp. 334 + ix, with numerous illustrations. 1946. Philadelphia and London: J. B. Lippincott Co. 24s. net.

The Surgery of Repair. Injuries and Burns. By Wing-Commander D. N. MATTHEWS, O.B.E., R.A.F.V.R. Second edition. $8\frac{3}{4} \times 5\frac{1}{2}$ in. Pp. 401 + xii, with 198 illustrations, many in colour. 1946. Oxford: Blackwell Scientific Publications Ltd. 45s. net.

Surgical Treatment of the Motor-Skeletal System. By 43 authors. Supervising Editor: FREDERIC W. BANCROFT, A.B., M.D., F.A.C.S., Associate Clinical Professor of Surgery, Columbia University; etc. Associate Editor: CLAY RAY MURRAY, M.D., F.A.C.S., Professor of Orthopedic Surgery, College of Physicians and Surgeons, Columbia University; etc. In two volumes. 10 × $6\frac{7}{8}$ in. Vol. I: Deformities, Paralytic Disorders, Muscles, Tendons, Bursæ, New Growths, Bones, Joints, Amputations. Pp. 641 + xix, with 521 illustrations. Vol. II: Fractures, Dislocations, Sprains, Muscle and Tendon Injuries, Birth Injuries, Military Surgery. Pp. 671 × xix, with 543 illustrations. 1946. Philadelphia and London: J. B. Lippincott Co. £6.

Amputation Prosthesis. Anatomic and Physiologic Considerations, with Principles of Alignment and Fitting designed for the Surgeon and Limb Manufacturer. By ATHA THOMAS, M.D., F.A.C.S., Associate Professor of Surgery (Orthopedics), University of Colorado School of Medicine, and CHESTER C. HADDAR, President of the Association of Limb Manufacturers of America. 9 × 6 in. Pp. 305 + xii, with 207 illustrations. 1946. Philadelphia and London: J. B. Lippincott Co. 50s. net.

Practical Points in Penicillin Treatment. By G. E. BEAUMONT, D. M. (Oxon.), F.R.C.P. (Lond.), Physician to the Middlesex Hospital, and K. N. V. PALMER, M.B. (Cantab.), M.R.C.P. (Lond.), Acting Medical Registrar to the Middlesex Hospital. $7\frac{1}{4} \times 4\frac{7}{8}$ in. Pp. 16 + iv, with 1 illustration. 1946. London: J. & A. Churchill. 1s. 6d. net.

Thromboses arterielles. Physiologie pathologique et Traitement chirurgical. By RENE LERICHE, Professeur au Collège de France, with the collaboration of D'IVAN BERTRAND, Directeur de l'Institut de Neurobiologie à la Salpêtrière. 9 × $5\frac{1}{2}$ in. Pp. 537 + viii, with 59 illustrations. 1946. Paris: Masson et Cie. Fr. 600.

Le Fibro-myome utérin. By J. DUCUING, Professor of Clinical Surgery at the Faculty of Medicine of Toulouse; Director of the Anti-cancer Centre. $9\frac{3}{4} \times 6\frac{3}{8}$ in. Pp. 541, with 156 illustrations. 1946. Paris: Masson et Cie. Fr. 735.